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Dr Graham Steell
with kind regards.
A. J.

ROYAL INFIRMARY CLINIQUES.

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ROYAL INFIRMARY CLINIQUES.

BY

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PREFATORY NOTE.

I HAVE long held the opinion that note-taking during Clinical Lectures has certain disadvantages associated with it, and it has been my custom, since I obtained Wards in the Edinburgh Royal Infirmary, to publish the various Clinical Lectures which it has been my duty to deliver. I am thus aware that most of these Lectures are already within the reach of those for whom they were intended,—namely, the students of the Class of Clinical Medicine. As, however, it is possible that some of them have been published in journals to which the students have not been able readily to gain access, I have thought it best to bring them together in this form.

To the students of the Clinical Medicine Class I have now much pleasure in dedicating this volume, in the hope that they may find its perusal of some value, and as a token of gratitude to them for their valuable assistance in obtaining the material out of which the various lectures were composed.

ALEX. JAMES, M.D.

10 MELVILLE CRESCENT,
June 1896.

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TWO CASES OF EMPYEMA.

GENTLEMEN,—We have had in our Ward examples of empyema arising in a variety of ways. We have had it following a sero-fibrinous effusion; we have had examples of it in which from the first the effusion seems to have been purulent; and we have had examples of it resulting from suppurative processes beneath the diaphragm. But we have had lately two examples of it occurring in a way in which it very often seems to occur, viz., associated with pneumonia, and to these I propose now to direct your attention.

The first is that of a man, Noah P., aged 56, a commercial traveller of Portuguese nationality, who was admitted to Ward 30, February 24, 1894, complaining of pain in the right side of his chest, of shortness of breath and great weakness, and stating that he had been ill six days.

Family History.—His mother died of consumption, and his father is also dead; but he has twelve brothers and sisters, all living and in good health, as far as he knows.

Personal History.—States he has always enjoyed good health. He travels about a good deal in his occupation, and so is exposed to all kinds of weather, but has always had good food and been well lodged. Up till a year ago he seems to have been a little inclined to alcoholic excess, but for the last year has been teetotal. He gives no history of any specific disease, or indeed of any previous serious illness.

Present Illness.—He states that six days before his admission he was feeling quite well. He had been indoors two

days because of the bad weather; but in the evening he had gone out for a walk, and on returning to his hotel he had felt very ill and had a severe shivering. He had then and for the following two or three days no great cough and no spit, but he felt a sharp pain in the right side of his chest when he drew a breath. On the fourth day of this illness he felt a little better, so that he ventured out for an hour or two, but on that same evening he felt distinctly worse,—the pain in the right side of the chest was very severe and was also felt in the abdomen. He had also a cough and was very feverish, so that he went to bed. Feeling himself getting worse and worse, he two days afterwards called in a doctor, who sent him to the Infirmary as suffering from pneumonia.

State on Admission.—Patient is a tall man, rather slightly built. Is evidently suffering from great pain in breathing. He is sweating profusely, and his face is rather cyanosed. His temperature is 100° F., his pulse is 104, respirations 32.

Respiratory System.—He complains of pain in the right side of his chest, as of a pin sticking into him and stopping his breathing, which is consequently shallow.

Physical examination of the lungs revealed over the right upper lobe anteriorly a tympanitic percussion note with harsh breathing and prolonged expiration. Over the right lower lobe posteriorly there was marked dulness of the percussion note, with somewhat faint bronchial breathing and a few crepitations. The vocal fremitus was noted to be very slightly marked, and the vocal resonance, though clear, was not loud. The expectoration was scanty, viscid, and rust coloured, and films of it stained showed the pneumococcus.

Circulatory System.—The pulse is about 104 per minute, and shows a tendency to irregularity as regards time and force of beat. It is rather soft and compressible, but the arterial walls are healthy considering the patient's age. The heart is in its normal position. Its sounds are rather weak,

but there is slight accentuation of the pulmonary second sound.

Digestive System.—The tongue is thickly furred. His appetite is gone, and there is great thirst. With the exception of evidence of slight splenic enlargement the abdominal viscera were found normal.

Urinary System.—The urine, about 40 ounces in the twenty-four hours, was rather high-coloured and slightly turbid. It contained a trace of albumen, but no blood and no sugar. There was slight diminution in the chlorides.

As regards the *Integumentary System*, it was noted that he sweated a great deal, especially on the head and chest; and as regards the *Nervous System*, it was noted that he was anxious and restless, and slept very little. He was treated by rest in bed, hot fomentations to the right side to relieve the pain, milk and beef-tea at short intervals, and for the condition of the heart, ammonia, strophanthus, and strychnine were administered at intervals.

His condition remained the same during the day after his admission, but on the second and third days, as a reference to the Chart will show, indications of some improvement were manifested. He stated that he felt better; his temperature and pulse-rate were lower, his cough was less painful, and the sputum more abundant, frothy, and muco-purulent. Still, his pulse was weak and tending to irregularity, and he looked ill, and the physical signs presented by his chest remained very much the same. In this state he remained until the 6th of March, when it was noticed that his temperature began to show oscillations. These oscillations continued and became more marked until the 9th of March, when the following was the note of his condition:—"Very slight improvement is manifested. The pain in the side is still present, but less than formerly; the pulse is still weak and tending to irregularity; the temperature rises at night, and with the fall in the morning temperature there is great

sweating. On examination of the chest, there is lessened vocal fremitus and faint tubular breathing over the right lower lobe, with a percussion note so absolutely dull as to suggest fluid, especially over an area slightly outside the inferior angle of the right scapula. Into this area a hypodermic needle was inserted, and some thin pus was found, which on staining revealed numerous pneumococci."

Now, gentlemen, we have in this case, I believe, an example of an empyema developing in a way in which it very often develops,—that is to say, associated with a pneumonia. In our patient we had, as you will have noted, a distinct history of a pneumonia. He had the initial rigor six days before his admission; he had the fever, pain in the side, cough, and later on the rusty, viscid spit containing the pneumococcus, the dyspnœa, the sweats, the slight albuminuria, and the other characteristic symptoms of the disease. Then he had a fairly marked crisis about the eighth day, as shown by the temperature chart, and corresponding with the fall in the temperature he described himself as feeling somewhat better.

But the question will occur to you—Was this a pure croupous pneumonia, and did the empyema develop upon it after the crisis, or was it from the first a pleuro-pneumonia?

Now here you will remember that every croupous pneumonia in which the inflammation has extended to the surface of the lung has associated with it a certain amount of pleurisy, but ordinarily such a pleurisy is accompanied by little or no fluid exudation. That this should be so you can easily understand when you remember that when a lower lobe is in a state of pneumonic consolidation there cannot be much space for fluid in the pleural cavity. But in our patient we believe that there has been a pleuro-pneumonia—*i.e.*, that there has been a croupous pneumonia of a part of the lower lobe of the right lung, and that an associated pleurisy had occurred and resulted in an effusion

Name Noah P. Age 56 Disease Empyema Result Recovery

FEB. 1894 | 24 | 25 | 26 | 27 | 28 | MAR 1 | 2 | 3 | 4 | 5 | 6 | 7 | 8 | 9 | 10 | 11 | 12 | 13 | 14 | 15 | 16

TEMPERATURE FAHRENHEIT'S SCALE

103° 102° 101° 100° 99° 98° 97° 96°

7th day of Pneumonia

Exploratory puncture pus found

Operation 19 oz. pus removed.

39° 38° 37° 36°

TEMPERATURE CENTIGRADE SCALE

Name James C. Age 7 Disease Empyema Result Recovery

APRIL 1894 | 16 | 17 | 18 | 19 | 20 | 21 | 22 | 23 | 24 | 25 | 26 | 27 | 28 | 29 | 30 | MAY 1 | 2 | 3 | 4 | 5 | 6

TEMPERATURE FAHRENHEIT'S SCALE

102° 101° 100° 99° 98° 97° 96°

Exploratory puncture pus found

Operation 6 oz pus removed

38° 37° 36°

TEMPERATURE CENTIGRADE SCALE

which had caused collapse of the remainder. That the pneumonia had been a pleuro-pneumonia was indicated, in the first place, by the physical signs. You will remember that over the dull lower lobe the vocal fremitus, though present, was not increased, as is usual in pneumonia; indeed, it was rather less marked than might have been expected, even with a healthy lung. Further, the tubular breathing sounded somewhat faint and distant, and the vocal resonance, though clear, was not intensified.

On the day on which the case was read (February 28) those peculiarities in the physical signs were noted, and whilst we contemplated the possibility of the presence of fluid, we remembered that such a condition might be the result of a blocking of some of the bronchial tubes with secretion. But, in the second place, the symptoms indicated a pleuro-pneumonia rather than a typical croupous pneumonia. Although the disease had begun with a rigor, and it showed a crisis on the eighth day, the crisis was not a complete one. Although the temperature fell below 99° F., and the pulse became slower, yet the patient was not well. He had still chest pain, and his pulse was weak and intermittent, so that strychnia, strophanthus, etc., had to be given. Now all this indicated that there was something more than an ordinary croupous pneumonia. And when about a week after the crisis the temperature chart began to show oscillations, we suspected empyema, and this was verified, as stated, on the 9th March by an exploratory puncture.

But now, granting that this had been a pleuro-pneumonia terminating in an empyema, do we know if the pleuritic fluid was purulent from the first? Or if the pleuritic fluid was not purulent from the first, can we say when it became so? These questions I cannot answer. I can say that in similar cases I have explored the chest with a hypodermic syringe, and obtained sero-fibrinous fluid, and a week or so afterwards I have punctured again and found pus. I can

say also that the oscillations of the temperature chart and the sweatings, so very characteristic of the presence of pus, seem to indicate resorption from a purulent collection, not a formation of pus.

The diagnosis having been made, the treatment was a simple matter. On the 10th March, chloroform being given and all antiseptic precautions observed, I made an incision into the right side below, and slightly external to the angle of the scapula, and let out 19 ounces of rather thin pus. An ordinary drainage-tube was inserted and dressings applied. After the operation there was very little sickness, and the patient expressed himself as much easier. On the day after the operation it was noted that the pulse was still weak and that the patient sweated a good deal, but that the temperature was down to normal. Rapid improvement set in from the second day after the operation, the temperature remained normal, the pulse improved in rate, strength, and regularity, the cough disappeared, the appetite improved, and the patient gained in strength. On the 22nd of March the drainage-tube was taken out, the discharge having ceased, and on the 23rd March (thirteen days after the operation) the patient got out of bed. On the 9th April the patient was discharged, recovered. The wound was healed, and there was only some slight dulness at the base of the right lung to be detected. All this will doubtless clear up in the course of a few weeks.

The next case to which I wish to direct your attention is that of the boy J. C., aged 7 years, who was admitted to Ward 30 on April 16, 1894.

We were told that he had been ill some five weeks, his illness having begun suddenly with fever and cough. Dr Macnab, who had recommended his admission, in writing me about him, stated—"I never at any period of his illness detected with certainty an effusion; all that I made out was a very distinct patch of pneumonia immediately in front of

his left scapula, which was a very considerable time in clearing up; perhaps it may not be entirely gone yet." (Six weeks after): "He had little cough during the day; at no time anything like distinct pneumonic spit." Dr Macnab further stated his suspicions of an empyema having occurred.

On admission we found the child pale and weakly-looking, with a very frequent and irritating cough. There was absolutely no expectoration,—but this, we had little difficulty in recognising, was simply because what should have been expectorated was swallowed. His pulse was rapid and feeble, but his temperature, though it showed some oscillations, was mostly subnormal. On examination of the chest we found at the left apex anteriorly a slight impairment of the percussion note, with harsh breathing and a few crepitations, and over the left lung posteriorly we found percussion impairment in the supra-spinous and upper interscapular regions, with prolonged expiration, harsh breathing, and some crepitation. At the base of the left lung, however, we found the percussion note more distinctly impaired, and the breath-sounds and vocal fremitus somewhat faint.

This was practically all that we could make out of the case, and the idea which first entered my mind to explain the condition was that there had been tubercular mischief at the left apex of some standing, and that some six weeks before, a basal pneumonia and pleurisy had occurred. In this connexion we had to regret that owing to the facts of the patient being too young to give any account of himself, and of his friends all residing in the country, we could not at the time ascertain whether the patient had had any symptoms of phthisis, cough, fever, night-sweats, etc., previously to his recent acute illness. Further, we could not get a specimen of spit to examine. The nurse exercised all her powers of persuasion to get the little patient to spit into the spittoon, but to no purpose.

I have now no doubt that had we succeeded in getting the

spit at that time, we should have made our diagnosis much earlier than we did,—for instead of the muco-purulent phthisical spit showing the tubercle bacilli, we should, I believe, have obtained the diffluent purulent spit characteristic of an empyema which is emptying itself, or attempting to do so, through the bronchi.

Partly because we contemplated the possibility of the lung mischief being tubercular, partly because the temperature continued a subnormal one, and partly because we continued to hope that we might yet get a specimen of the spit for examination, we, for nearly two weeks after his admission, limited our efforts to the endeavour to improve his general health. We gave him nourishing food at short intervals, with a teaspoonful of the Vin Nourry twice a day. During this time, however, absolutely no improvement showed itself; indeed, we did not fail to notice that his general condition and cough were becoming distinctly worse. On April 29 (thirteen days after his admission) it was noted that his temperature, hitherto subnormal, had begun to rise at night, and that the percussion note at the base of the left lung continued very dull. Further, that at a point near the angle of the left scapula the percussion note was specially "dead," and distinctly suggestive of the presence of fluid. I therefore made an exploratory puncture there, and finding pus, I next day, with all antiseptic precautions, incised the chest, evacuated 6 ozs. of pus, and inserted a drainage-tube.

The child's progress towards recovery was practically uninterrupted. The temperature and pulse-rate returned to normal, the irritating cough ceased, and the appetite, digestion, and general condition improved. By the middle of May the tube was removed and the patient was out of bed, and on June 2 he was discharged, recovered.

These two cases seem to me good examples of empyema associated with or following pneumonia. The first case was, from the diagnostic point of view, perfectly simple, but the

second was rather confusing. In it the physical signs were not at all distinctive, and the consideration of the temperature chart rather misled than helped us for a time. I believe that these anomalies, as regards physical signs and temperature, were mainly the result of the fact that the purulent collection was to some extent finding its way out through the bronchi. The quantity of pus in the chest was thus comparatively small, and the lung was adherent round about, so that vocal fremitus and breath-sounds were never absent. Further, there was for a time no resorption fever, which would have indicated on the temperature chart the presence of pus. Had the condition been in an adult, so that we might have been able to get the spit, we should probably have recognised it earlier. As already mentioned, in such cases the spit is purely purulent, and it runs together in the spittoon,—that is to say, the individual spits cannot be seen.

An important point, however, which all this brings out prominently, is that exploratory puncture is the only sure way of recognising empyema. I have said exploratory puncture—I should rather have said repeated exploratory punctures—for in cases of small localized empyemas it often happens that one does not strike the pus until after several attempts. For such punctures I am in the habit of using a rather large-sized needle, with at least one side opening, so that if the point goes through into the lung, the pus will still be drawn out by the side opening. You may ask, Does exploratory puncture ever do harm? To this I would answer, that I have never seen any harm result from it, but that I have often seen great harm result from its neglect.

PULSATING EMPYEMA.

GENTLEMEN,—I propose to bring before you to-day a case of pulsating empyema, and in connexion with it to discuss with you in some detail the cause of the pulsation.

T. R., aged 26, a brewer's clerk, was admitted to Ward 30 on January 15, 1895, complaining of pain over the heart, with swelling in the same region, and a cough. His father died at the age of 32 of heart disease. He had one brother who died at the age of 18 of heart disease, and a sister who is delicate and suffers from a cough. His mother, another brother, and two sisters are alive and healthy. The patient had measles when a child. Eight years ago he had to give up his occupation as pupil-teacher owing to nervous strain. He has had no accidents nor other illnesses until the present one.

His present illness seems to have commenced in July 1894. He then caught cold, and he noticed that on taking a deep breath or sneezing he felt a pain over the region of his heart. He still, however, continued at his work, and he expressly states that he had no cough and no night-sweats. About the 5th or 6th of January, however, he seems to have again caught cold. A cough then commenced, which has continued and which has been accompanied by a severe pain over the heart. A few days after he caught this cold he noticed a swelling over his heart. This and the cough and weakness, with shortness of breath on exertion, led him to consult a doctor, who recommended his coming to the Infirmary.

State on Admission.—Patient is about 5 feet 6 inches in height, and weighs 110½ pounds. He says he has been losing flesh lately. His temperature varies between 97° and 99°; his pulse is usually about 80 per minute.

Over the præcordia a pulsating expansile swelling is seen. It is somewhat diffuse, and in extent is about 4 inches in diameter, its centre being between the fourth and fifth ribs. On palpation, its expansile character is better recognised. It can be somewhat reduced by steady pressure, and when the patient coughs it can be felt markedly to increase in size. There is pain when its centre between the fourth and fifth ribs is pressed upon. Over the swelling, and over the lower half of the sternum, there is some slight œdema.

Percussion gives complete dulness over the whole of this swollen area, and over the entire upper lobe of the left lung in front there is a markedly impaired note. Over the lower and lateral portion of this lung the percussion note is, however, resonant. Posteriorly there is impairment on percussion, from the apex down to nearly the angle of the left scapula. Over those parts of the lung which are dull to percussion, auscultation reveals weak bronchial breathing. At the apex of the left side, anteriorly and posteriorly, a few crepitations can be heard. Over the lower part of the left lung, and over the whole of the right lung, the breath-sounds are vesicular in character; over the right lung they are, in addition, exaggerated. Over the swelling in the præcordia, and the neighbouring portions of the left upper lobe in front, vocal fremitus is absent. Over the rest of the left lung it can be detected.

The precise position of the heart cannot be made out exactly by percussion, but it seems to be displaced to some extent to the right. With the exception of a somewhat reduplicated first sound, its sounds are normal.

The other systems and organs are healthy.

In making a diagnosis in this case, several possibilities entered our minds. There was, in the first place, the possibility of aneurism, which, indeed, we were informed, had been suspected prior to his admission. But we remembered that our patient gave a history of lung trouble; that the pulsating tumour had shown itself suddenly, and had rapidly reached its present stage. We noted, further, that the pulsating swelling was not in the position usual in cases of aneurism; that there was no evidence of cardiac or vascular disease, and that there were no pressure symptoms of aneurism. Finally, we had no hesitation in making an exploratory puncture with a hypodermic syringe, and we readily ascertained the presence of pus. Next we thought of the possibility of suppuration from a diseased rib or from mediastinal abscess. As regards the former supposition, we remembered that in such cases the pus is more apt to come outward than to extend into the pleural cavity; whilst as regards the latter supposition,—viz., mediastinal abscess,—we remembered that such a condition would not likely cause such an extensive area of dulness over the upper part of the left lung as was present in this case. Then we naturally thought of pulsating empyema, and we noted that in some ways the symptoms and physical signs seemed to indicate, and in other ways to contraindicate this. As regards symptoms, there had been no marked cough, and there was practically no fever, hectic, nor sweatings, such as one expects to find in empyema. But, on the other hand, these symptoms are often absent, and we remembered that in pulsating empyemas the symptoms are apt to be latent in a special degree. Then, as regards physical signs, we found that the empyema, if it were such, was limited to the upper part of the left lung, and this, of course, we recognised as very unusual. But, taking into consideration the history of lung-trouble of some months' duration, and the fact that there was evidently apical disease, as betokened by the

crepitation and other physical signs, we came to the conclusion—(1) that our patient had been suffering for at least several months from tubercular disease of the left apex; (2) that this had resulted in pleuritic adhesions over various parts of the left upper lobe; (3) that over a portion of this lobe anteriorly, where adhesions had not formed, a purulent pleurisy had started; (4) that the pus from this, prevented from gravitating to the lower part of the pleural cavity by adhesions, had collected in the upper part, and had found its way outward between the fourth and fifth ribs, thus forming the pulsating swelling over the præcordia. This being our diagnosis, the treatment required was obvious. The patient having been chloroformed, an incision was made over and into the pulsating swelling. This abscess cavity having been opened, the finger introduced through the incision found a large opening of communication between it and the empyema cavity. This was found to be large enough to contain some fourteen ounces of pus, and, as had been diagnosed, to be limited to the upper part of the side. In order to facilitate drainage, an opening was made into this cavity between the fifth and sixth ribs in the anterior axillary line, a small portion of rib resected, and an ordinary drainage-tube inserted. With the removal of the pus it was specially noticed that the heart, slightly displaced to the right, showed no tendency to resume its normal position. All antiseptic precautions were, of course, observed at the operation, and have been at the subsequent dressings.

The progress of this case I shall refer to later on; in the meantime I propose to go over with you in some detail the subject of pulsating empyema. Of this, two forms are to be recognised,—(1) pulsating intrapleural empyema, and (2) pulsating empyema of necessity. In the former, all the fluid is in the pleural cavity; in the latter, a certain amount of the fluid has, as in our patient, found its way outside of the pleural cavity, and forms under the skin a pulsating

tumour. In both cases the left is the side almost invariably affected, and the effusion is usually at the lower part of the chest,—not, as in our patient, localized in the upper portion.

Pulsating Intrapleural Empyema.—Observe, in the first place, that all the pulsating intrapleural effusions which have been reported have not been purulent. Several instances of pulsating sero-fibrinous pleuritic collections have been met with, and interesting examples of such have been given, among others, by Douglas Powell and Comby. But the great majority of these cases have been purulent from the first, or have become so in the course of the disease. Hence we are justified in speaking of them as pulsating empyemas. Some of the earliest recorded cases of this are those of Macdonell[1] and Stokes.[2] Stokes's case is especially interesting. He describes how, in a huge empyema, which had powerfully pushed the heart to the right, he had observed pulsation of the entire sac. "Thoracocentesis was performed three times, and each time a large quantity of more and more purulent fluid was removed. Before each operation the beats of the heart produced a diastolic (?) pulsation of a most strange character, which made itself felt in the left lateral portion of the chest and through the entire thorax. The bed was shaken by each contraction of the heart, the force of which seemed to be increased,—the violence and extent of the pulsations were such that the sleep of the patient was interrupted. The fluid had never any tendency to find its way outside, and it is very remarkable that the heart never left its position to the right of the sternum after any of the emptyings of the chest." Other more or less similar cases have been reported, the most complete collection of which I believe to be that given in Comby's recent work, *L'Empyème Pulsatile,* to which I would refer you.

Pulsating Empyema of Necessity.—In this form, as already

[1] *Dublin Journal of Medical Science,* March 1844.

[2] *Diseases of the Heart and Aorta.* Dublin, 1854.

stated, the fluid has found its way through the chest parietes, and forms a subcutaneous tumour, pulsating and expansile, at some part outside of the thoracic cavity. Like the former variety, it is almost invariably in the left side of the chest, and the fluid is usually at its lower part. Unlike the former variety, however, the fluid is always purulent. This can easily be understood when we remember that only a purulent fluid can penetrate the chest-wall. Further, the pleuritic process which has given rise to this effusion is usually a chronic one. This we can understand when we remember that time is required for the accomplishment of the process of perforation. As regards the position of the pulsating tumours, this is most frequently found, as in our patient, to be about the præcordial region,—that is to say, on the left side, somewhere between the mid-sternal and nipple lines. But just as an empyema may perforate the chest-wall and point elsewhere, so the pulsating tumour may be found elsewhere,—*e.g.*, in the posterior or lateral parts of the chest, or even in the lumbar region. These tumours vary in size, they are usually, as in our patient, somewhere about 4 inches in diameter, and the expansile character of the pulsation can usually easily be recognised. The respiratory act readily affects such tumours. As can be understood, whilst inspiration is apt to cause a slight diminution, expiration may cause a corresponding increase in their size. The acts of coughing and sneezing are specially apt to increase their bulk, and a change may also be produced by the patient's sitting up or lying down. The tumour may to some extent be reducible, and cases have been reported where partial reduction by pressure and manipulation caused a marked increase in the dyspnœa. It is evident that to permit of the occurrence of these variations in the size of the tumour, the opening of communication between it and the intrapleural collection must be free. In our patient this opening readily admitted the finger.

But now, how is the occurrence of pulsating empyema either with or without an extrapleural tumour to be explained? It is evident, in the first place, that the condition or conditions which give rise to this must be rare. Cases of large pleuritic effusion and cases of empyema necessitatis are not uncommon, and yet the pulsating character is very rarely met with. What, then, is it which causes the pulsation?

On this subject a great many theories have been brought forward, to some of which we may refer. Let me first quote the following from Guéneau de Mussy, "Quand avec l'épanchement se trouve, dans la cavité thoracique, un gaz éminemment compressible ou une portion considérable du poumon encore perméable à l'air, on comprend que la systole ventriculaire puisse refouler le liquide contigu au péricarde et lui communiquer un ébranlement ondulatoire qu'elle ne peut pas produire quand elle agit sur une masse absolument incompressible."

Féréol seems to have practically adopted this, and bases his explanation of the occurrence of pulsation upon the existence of an associated pneumothorax. But cases have frequently occurred where there was no pneumothorax: our patient is an example of such.

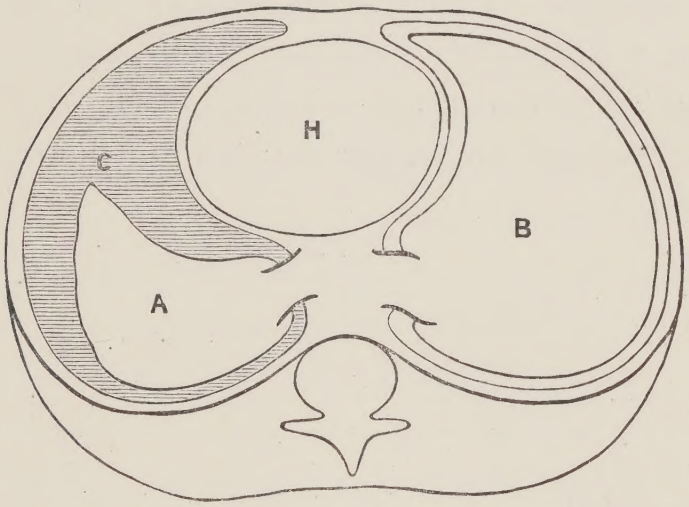
Traube seems to have believed that the pulsation depended in some way on pericarditis and pericardial effusion, but cases have occurred without these. Walshe[1] says of the pulsation that it "seems to be merely an excess of the slight fluctuation movement in the fluid which is not very uncommon in ordinary cases." Powell[2] concludes that "probably nothing more is needed than an amount of fluid which shall exercise a certain degree of pressure, neither too much nor too little, upon the beating heart." Many other explanations have been proposed, for details of which I

[1] *Diseases of the Lungs.* London, 1871.

[2] *Diseases of the Lungs and Pleuræ.* London, 1886.

would refer you to the works of Bouveret[1] and Comby,[2] but, as it seems to me that none of them are quite satisfactory, and that a study of the physical conditions existing in the thorax may help us in this matter, I shall briefly proceed to this.

FIG. 1.



Section of thorax at level of sixth dorsal vertebra in a case of left-sided pleural effusion.

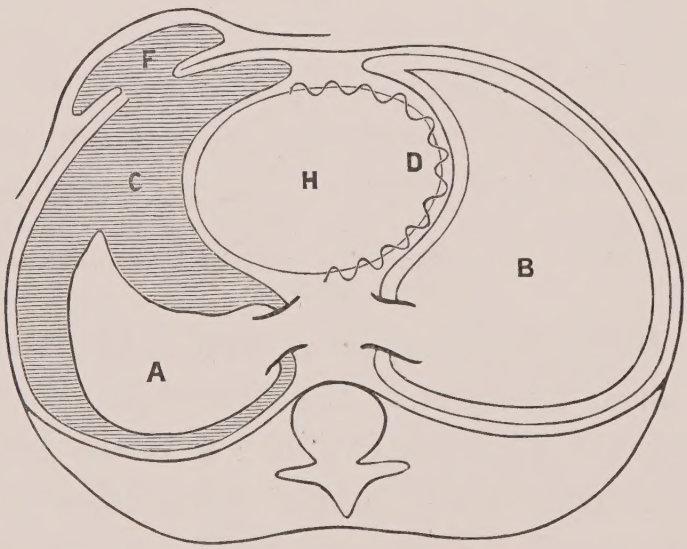
Let the Diagram (Fig. 1) represent a section through the thorax at about the level of the sixth dorsal vertebra in a patient with left pleuritic effusion: H is the heart, A the collapsed left lung, B the healthy right lung, and C the effusion. It is evident that as the effusion is, like all fluids, practically incompressible, and as it is contained in a cavity the walls of which are practically rigid and unyielding, the systolic and diastolic movements of the heart will be completely resisted on this side. On the other hand, they will readily be allowed to take place on the right side, the healthy and elastic air-containing right lung readily yielding to any enlargement of, or movement of, the heart to the right, and as readily following any diminution of, or movement of, the heart to the left. But now supposing that along the zigzag line D (see Fig. 2) the heart be fixed, by pleuritic, pericardial,

[1] *L'Empyème*, Paris, 1888.

[2] *L'Empyème Pulsatile*, Paris, 1895.

or mediastinal adhesions, and supposing that the fluid C in the left pleural cavity be in free communication with a subcutaneous abscess cavity F, it is evident that the mechanical conditions will be quite reversed. Increase or diminution in the size of the heart, or movement of any part of it to right or left in systole or diastole, will readily affect the fluid effusion. This can be because the fluid in the pleural cavity will readily yield and recoil with the cardiac movements, being now in free communication with the fluid in the subcutaneous cavity, the walls of which are distensile and elastic.

FIG. 2.



Section of thorax at same level. At D adhesions between the pericardium and pleura have occurred, followed by bulging of the effusion through the chest-wall.

Hence, we should conclude that in pulsating empyema of necessity the factors which bring about the pulsation are (1) fixation of the heart, and (2) distensibility and elasticity of the walls of the empyema sac.

But it will now be asked, How is the existence of pulsation in intrapleural empyemas or in other intrapleural fluid collections to be explained? To this I would answer that the factors are practically the same. The heart must be more or less fixed, as in the former case, whilst the yielding and recoiling of the fluid to its movements may be due to

such conditions as the presence of air in the pleural cavity, to the existence of a portion of semi-inflated lung, to a yielding and elastic diaphragm, to the yielding of the interspaces through paralysis of the intercostal muscles, or even to the comparatively yielding chest parietes of early life. Perhaps the best illustration of how pulsation may be brought about by yielding parietes is to be obtained from a study of the cranial circulation. Thus if in the cranium of a living animal a trephine opening be made, through this opening the exposed brain can readily be seen to pulsate. If now this opening be completely closed by a piece of glass, it will at once be seen that the pulsations cease. Hence, we may say that whatever permits of the yielding of walls tends to bring about pulsation.

It is evident that the degree of fixation of the heart, and the degree of yielding and recoil which the chest parietes or contents permit to the fluid, must vary much in different cases. Thus the strength and extent of the pulsations in pulsating empyemas must vary correspondingly. In Stokes's case, previously referred to, we are told that the strength of the pulsations was so great as to shake the patient's bed. This statement has been regarded by some as incredible; but we have little hesitation in believing it, if we remember the hydrostatic law, and the fact that the normal contractile force of the heart is somewhere about 4 pounds per square inch. We can understand again, in other cases, how the pulsations may be so slight as to be hardly perceptible, and we cannot doubt that, if carefully looked for, pulsation might be found in many cases in which its existence has not been suspected.

And now to refer again to our patient. Since the removal of the pus he has felt considerable relief, the left lung is expanding, and the heart is showing a tendency to return to its normal position. But he is now evincing the symptoms of phthisis, he has a muco-purulent spit containing the

tubercle bacillus, and his temperature chart and pulse-rate betoken fever. Whilst we may anticipate for him some immediate relief from our interference, our ultimate prognosis is of course a very grave one.

[This prognosis was borne out by events. Up till the end of February he remained fairly well, although indications of slight sero-fibrinous effusions at the left base had shown themselves. By the beginning of March, however, he was evidently losing ground, as the following excerpts from the case-book show:—

March 8.—Patient is being dressed every other day; the discharge is but slight. Temperature has begun to show oscillations, rising to 102° at night. Appetite and general condition not so well maintained.

March 13.—Temperature as before; general condition of patient not so good, more easily wearied, and appetite failing.

March 20.—Complains of headache, and towards evening became somewhat delirious, trying to get out of bed. Tubercular meningitis suspected.

March 22.—Headache persistent, and does not recognise his friends. Is refusing food.

March 23.—Condition worse; pupils react very sluggishly.

March 24.—Practically comatose.

March 25.—Died this evening.

Permission to examine the chest only was obtained, and the following is Dr Muir's report:—

"*Sectio*, March 28, 1895.

"*External Appearances.*—Body was much emaciated, and there was marked pallor of the surface. Rigidity slight, lividity slight posteriorly. There was an opening in the left side into the pleura in the fourth space close to the sternum; another further back where a portion of the fifth rib had been resected.

"*Thorax.*—Slight adhesions on the inner aspect of the right

lung. Pericardium normal. In the left pleura there was an old empyema over the upper anterior part of the lung. The cavity contained a small quantity of pus, and was limited by dense fibrous adhesions. There was great fibrous thickening along the left border of the sternum, fixing the pericardial sac. Over the rest of the lung there were scattered fibrous adhesions, and there was a small quantity of serous effusion at the base posteriorly.

"*Left Lung.*—Weight, 1 lb. 12 ozs. The pleura showed great thickening in relation to the empyema cavity. There were one or two old tubercular nodules scattered through the upper lobe with fibrous thickening around, but there were no cavities. Throughout the lung there was well-marked tubercle, the nodules being fairly numerous, grey in colour, and about the size of a pin's head.

"*Right Lung.*—Weight, 1 lb. 11 ozs. It was congested posteriorly, and showed acute tuberculosis, as in the other.

"*Heart.*—Weight, 10 ozs. All the valves were normal, cavities of normal size, myocardium pale and soft, not fatty but anæmic."]

ON A

CASE OF FIBROID PHTHISIS, WITH GREAT DISPLACEMENT OF THE HEART.

GENTLEMEN,—J. S., aged 52, a pilot, was admitted to Ward 30, October 14, 1892, complaining of pain in the side and cough.

History.—An uncle on his mother's side died of stone-mason's phthisis, but with this exception his family history is excellent. He is the father of eight children, of whom six are well and strong; the other two died in infancy. Habits have always been temperate, and his surroundings at home have always been favourable. At work he is somewhat exposed, but as a pilot his work is not unhealthy.

Previous Illnesses and Accidents.—Twenty-two years ago, when sitting on the top of a loaded omnibus with his back to the horses, he was driven through an archway which seems to have been too low for its requirements, for the back of his head was knocked against it so violently that he was thrown senseless to the ground. He soon recovered from this, but he states that ever since then his sense of smell has been completely lost. Ten years ago, when pulling in a boat race, he became completely exhausted, and had to be lifted out of the boat. He then threw up more than a large teacupful of blood, and for about two months continued to throw up blood in smaller quantities. Ever since then he states that

he has had a cough, but up till two years ago it troubled him so little that he paid no attention to it. Two years ago, after exposure to bad weather, he complained of pain in his left side, and for this he came to the Infirmary, and was treated for a dry pleurisy. After six weeks' treatment he went out much improved. At this time the condition of his right lung and the position of his heart, to which we shall have to refer later, were observed.

Present Illness.—Six weeks before his admission he had a severe bleeding—throwing up at least a breakfast-cupful of pure blood. This bleeding, but in smaller quantity, continued for nearly a fortnight, and he states that during this period he also spat up material "as from a rotten gathering." Four weeks ago a severe, cutting pain arose on his left side at the same place as two years ago, and finding himself unable to work, he came again to the Infirmary. Although his present illness began six weeks ago, he seems to have been distinctly weaker for over a year. His appetite has been failing: for example, after a small breakfast he rarely had more than a sandwich and a glass of whisky—and often not caring for this—till night, when at supper a cup of tea and bread, with perhaps a little fish, sufficed.

State on Admission.—Height, 5 ft. $4\frac{3}{4}$ in.; weight, 7 st. $6\frac{3}{4}$ lbs.; before this illness was 9 st. 6 lbs. His muscles have the appearance of having been well developed, but are now small and flabby. Temperature varies from 98° to 99° F.; pulse about 70 per min.

Respiratory System.—Respirations, 25 per min. Cough slight, specially in the mornings. Spit scanty, viscid, muco-purulent and opaque. Repeated examinations have been made for bacilli, and we have concluded that, if these or lung tissue are present at all, they must be in very minute quantity.

On inspection and palpation, marked flattening is recognised over the upper part of the right chest, with deficient respiratory movement and increased vocal fremitus. Over

the second and third right interspaces anteriorly the palpating hand can feel crepitations. The entire left chest seems enlarged. On measurement over the nipples the left side is found to be $16\frac{1}{2}$ inches as against $16\frac{1}{4}$ on the right side. It appears, therefore, to be larger than it really is, but it is to be remembered that the right chest is normally the larger.

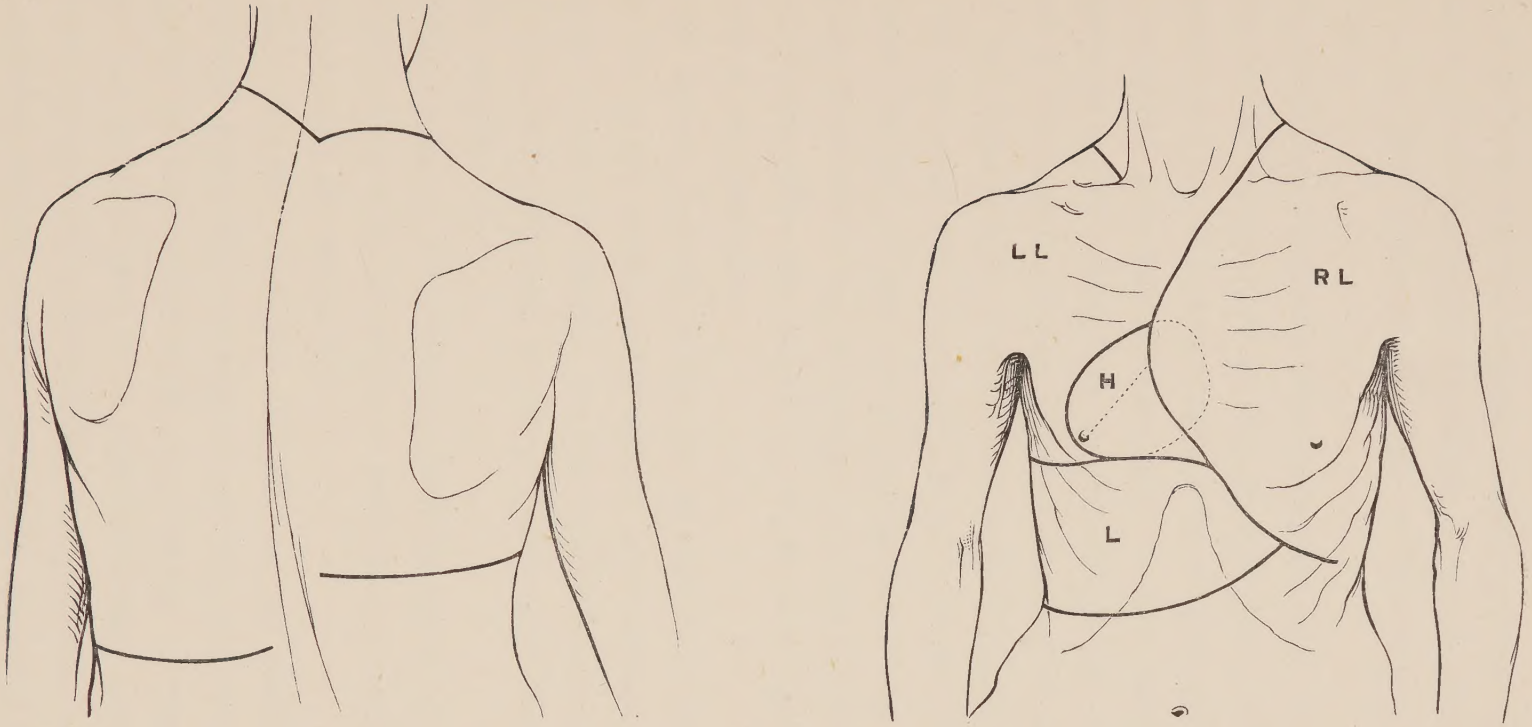
On percussion, dulness is found over the upper part of the right lung, extending downwards to the fourth rib anteriorly, and to about 1 in. below the spine of the scapula posteriorly. Over this dull area, but specially marked in the infra-clavicular region, auscultation reveals prolongation of the expiration—bronchial or cavernous breathing—coarse consonating crepitation, and increased vocal resonance and whispering pectoriloquy. Over the lower portions of this lung the breathing is harsh and indeterminate, with a few crepitations and rhonchi. Over the left lung the percussion note is everywhere resonant, and the breathing is puerile, with here and there a few rhonchi and crepitations, and in the sixth and seventh interspaces in the axillary line well-marked friction is heard.

By percussion and auscultation the boundaries of the lungs are made out as follows (see Plate):—

Left Lung.—Upper border can be traced from the left sterno-clavicular articulation upwards and outwards to the edge of the trapezius muscle, where it is $2\frac{1}{4}$ in. above the level of the clavicle, thence inwards and downwards to the vertebra prominens.

Right Lung.—Upper border cannot well be demonstrated at the right sterno-clavicular articulation, but at the anterior edge of the trapezius muscle is $1\frac{1}{4}$ in. above the level of the clavicle. From this point it can be traced to the vertebra prominens along a line which is nearly straight.

Left Lung.—Anterior border can be traced from the left sterno-clavicular articulation to the right and downwards, so



The above Figures (from photographs) show the shrinking of the right lung, the hypertrophous emphysema of the left, and the displacement of the heart and liver.

that at the second right interspace it reaches a point one-half inch to the right of the right border of the sternum. Thence it passes downwards, slightly inclining to the left, till at the level of the fifth costal cartilage it meets the liver dulness about one-half inch to the left of the right border of the sternum.

Right Lung.—Anterior border cannot well be traced above, owing to its being more or less consolidated, but, in the right parasternal line, is found at the level of the second interspace an impaired percussion note, and at the level of the third interspace a dull note, due to the displaced heart. At the level of the fourth rib the anterior border of the right lung is at the right nipple, being apparently retained at this point by the apex of the displaced heart. From this point the anterior border of the lung passes obliquely inwards and slightly downwards till it merges into the liver dulness at the level of the fifth rib, about 1¼ in. to the right of the right border of the sternum.

Left Lung.—Lower border in parasternal line corresponds with the sixth costal cartilage, in the mammillary with the seventh costal cartilage. In the axillary line it corresponds with the ninth rib, and in the scapular with the lower border of the twelfth rib.

Right Lung.—Lower border corresponds in the mammillary line with the fifth, in axillary with the seventh, and in the scapular with the ninth rib.

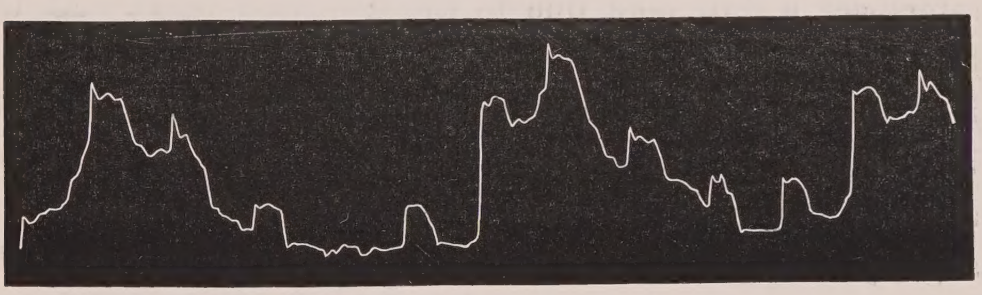
These marked changes are all, as we shall see shortly, connected with the cicatricial healing processes which are going on in connexion with the disease in the right upper lobe. Let us now pass on to the circulatory system.

Pulse, 72 per min., beat rather soft; arterial walls normal.

Inspection shows visible pulsation in second, third, and fourth right interspaces, and a very distinct impulse beat in the fourth interspace about half an inch internal to right nipple line. These signs are corroborated by palpation.

Percussion shows the dull area of the heart to be irregularly quadrangular in shape, its extreme right limit being at the right nipple, its extreme left about the middle of the sternum, its upper border coursing obliquely across the second and third interspaces, and its lower border merging into the liver.

Auscultation shows nothing more than that the heart sounds are heard loudest to the right of the sternum, and a cardiographic tracing taken from the impulse beat in the fourth right interspace has all the characters of an ordinary apex tracing (see Tracing).



Digestive System.—Though his appetite is not great, his digestion seems to be fairly well performed. He is always rather constipated. Inspection and palpation of the abdomen reveal nothing abnormal. The liver is evidently drawn upwards, its upper border corresponding in the nipple, axillary, and scapular lines with the fifth, seventh, and ninth ribs, its lower border being about 1 in. higher than the costal margin in the nipple line. The spleen appears normal.

Urinary System.—Amount 40 ozs., acid ; sp. gr., 1020; no albumen.

Integumentary System.—Normal; never perspires much.

Nervous System.—Normal, with the exception of his loss of the sense of smell.

The diagnosis made was chronic pneumonic or fibroid phthisis of the upper lobe of the right lung of some ten years' duration. Though gradually progressive, it has been associated with, and its progress restrained by, marked cicatricial fibrosis of the lung tissue, and this fibrosis had led to the shrinking downwards of the lung apex, to the shrinking

upwards of the lung base, to the marked displacement of the heart to the right, and to the enlargement (hypertrophic emphysema) and dragging over of the sound lung.

Now I wish to say something about this fibrosis and its results in general, and in this case in particular. Observe, in the first place, that in the main it is a salutary process. Occurring around foci of softening or around cavities, it tends, like cicatricial tissue elsewhere, to contract and encapsule or obliterate them, and in many cases where the lung mischief is limited it completely succeeds in its healing efforts. Unfortunately in so many cases the foci of disease are multiple, and so contraction around one focus or set of foci is apt to drag on or tear the new-formed tissue round others, to dilate other cavities or bronchi, and so to act injuriously. Like everything else in nature, this fibrosis presents harmful as well as beneficial aspects, but like everything else in nature it tends also in the main towards the latter; and where, as in our patient, the yielding of the parts around—chest-wall, liver, healthy portions of lung, heart, and opposite lung—can all work together to help it, it is wonderful how the effects of disease can by it be counteracted.

This patient illustrates the immense importance, from the prognostic point of view, of the disease being unilateral. It is evident that if the other apex, the left, were also affected, not only would the fibrotic change in connexion with it be rendered more difficult, but it could not yield the same assistance to the disease at the right apex. The most rapid cases of phthisis are those in which at an early stage both apices are found affected, and in practice you will often find that a patient in whom unilateral disease has gone on till the lung is little more than a mass of fibrous tissue and cavities, will come to see you year after year, looking little the worse; whilst another, in whom you have found only a little impaired percussion note and crackling at both apices, has lived only a few months.

The displacement of the heart to the right which this patient presents is more than one usually sees, and merits a few remarks. I have seen it occurring to a similar extent in four cases—three males and one female. In two of the males the disease of the lung which had brought it about had been arrested years before I had seen them, and coming before me for other ailments, the condition of the heart had been recognised accidentally. The evidence of old lung mischief was easily made out in the shape of impaired percussion note and weak respiratory murmur at the right apex, and indications of shrinking in this locality. In three of the male cases (including the patient whose case we are now considering) there was a distinct history of strain.

The mode of displacement of the heart in this and other such cases I believe to be as follows:—With the base of the heart as the fixed point and centre of a circle, the long axis of the heart, like the radius, has described an arc to the right, whilst at the same time the organ has rotated on its long axis so as to bring the left ventricle more anterior. No doubt the entire organ and the mediastinum are dragged over to some extent *en bloc*, but I believe that this tilting of the apex to the right and the rotation are the chief causes; and it is to be remembered, too, that the dragging up of the liver may now be assisting in this displacement. With such a displacement one might suppose that some twisting or kinking of the large vessels would certainly occur and cause bruits, but such bruits have not markedly been present in the examples of this condition which I have seen.

A question which you may ask here is, How long has this displacement existed? We cannot say definitely. We know that it has been so for the last two years at least; we may suppose that it has been so for a much longer time. In one of the other cases, in which I found a very similar displacement, the mischief in the right upper lobe had existed for two years.

So much for the fibrosis and the results of it which this patient presents; let us now say a little as regards some other features in his case.

It will be remembered that the disease seems to have begun some ten years ago, when as the result of a severe strain he felt faint and coughed up a large quantity of blood. He tells us that since then he has noticed the cough, which is still going on. Are we to suppose that this is a case of *phthisis ab hæmoptoe*, that we have here an example of a hæmorrhage setting up a chronic phthisical process? Or are we to suppose that, unknown to our patient, there had been some tubercular deposit at his right apex in connexion with and eroding the wall of an artery; and that, as the result of the increased blood-pressure which the straining entailed, the wall had given way at that point and caused the hæmorrhage? By the older writers the first view would have been upheld, whilst the new school would most strenuously uphold the latter. To say which has been the cause is well-nigh impossible. Although his family history is good, although his health prior to the accident had been excellent, and although subsequent to his illness his constitutional strength has been able so well to counteract the ravage of the lung disease, we can never be sure that tubercular deposit did not pre-exist. Further, we know that in the great majority of cases of phthisis which are said to have begun by a hæmorrhage, closer inquiry will show that tubercular deposit and erosion of a vessel has been the real factor. On the other hand, we cannot deny that *phthisis ab hæmoptoe* may have a real existence. Hæmorrhage into the lung may occur independently of tubercle, and hæmorrhage means injury and irritation of the lung tissue, and the induction in it of a nutritive condition so weakened that the attacks of the ubiquitous tubercle bacilli cannot be withstood. The precise meaning of the first hæmorrhage in this case cannot well be understood. Had it been caused by some external injury

instead of by a strain, the argument in favour of the *phthisis ab hæmoptoe* theory would, of course, have been stronger.[1]

But next it is to be noted that subsequent bleedings have occurred. These, as regards causation, are easily understood. By the fibrotic contraction cavities and dilated bronchi have their walls dragged upon here and there, and arteries in these walls exposed. These, from want of support and other causes, develop miliary aneurisms, which in their turn are very liable to rupture. Indeed, it is in cases of this kind, with fibrosis, cavities, and dilated bronchi, that severe and sometimes fatal hæmorrhages are most prone to occur. These are the cases in which miliary aneurisms are most common, and the patient's general condition being fairly good, the heart is strong and the blood-pressure high.

Next, as regards prognosis in this case. There is no doubt that in such patients the immediate prognosis is favourable enough. In the absence of active lung mischief the cough and expectoration cause little trouble, whilst fever, with its associated anorexia, dyspeptic troubles, thirst, sweatings, etc., is absent.

But the remote prognosis is most unfavourable; the extent of the disease has long since precluded the possibility of complete arrest and healing. The cough and expectoration must continue, and as time goes on we may expect that, with the gradual lessening of the nutritive power, the disease will spread more rapidly and the fibrotic contraction will be less efficient to counteract it. A tubercular element may gain prominence. The other lung may be involved, and this, as we have described, would tremendously accelerate the progress of the disease, whilst attacks of hæmorrhage are, as we have seen, likely to occur from time to time, and still more weaken the patient.

With the lessening of the ærating surface in the lungs,

[1] For instances of phthisis following injury to the lung, see Mendelsohn, *Zeitschrift fiir Klin. Medicin*, Bd. 10.

congestion of these organs will be apt to occur, and this will entail increased liability to attacks of bronchitis, or pleurisy, or pneumonia. Further, the backward pressure of the blood is apt to extend to the right heart and cavæ, and with ensuing congestion of the liver and portal system, of the kidney and of the lower limbs, digestive troubles, nephritic troubles, and dropsy may at any time show themselves. In such cases clubbed fingers are distinctly frequent.

The treatment in instances of this kind is mainly attendance to the general health and the careful avoidance of cold. Cough mixtures are quite unnecessary,—a warm drink—tea or milk—in the morning being all that is required for the cough and expectoration. The diet should be as nourishing and in as small bulk as possible, and should therefore consist to a large extent of animal food; alcohol in one or other of its forms has been recommended on the theory that it promotes the formation of fibrous tissue. Cod-liver oil and syrup of iodide of iron seem often to suit such patients, whilst any work or exercise which necessitates strain should for obvious reasons be avoided. Sunlight and the open air are always to be desired, and were our patient's circumstances such as to permit of his spending the winter and spring months under good climatic conditions, he might without doubt even yet add years to his life.

The only other point in this case to which I wish to refer is the loss of the sense of smell. This I believe to have been due to the blow on the back of his head (see page 22) having caused rupture and consequent degeneration of the fibres of the olfactory nerve at their points of passage through the cribriform plate of the ethmoid bone.

ON A

CASE OF RENAL AND CARDIAC DISEASE,

WITH SPECIAL REFERENCE TO THE DIURNAL AND NOCTURNAL EXCRETION OF URINE.

GENTLEMEN,—J. W., æt. 39, a blacksmith, residing in Edinburgh, was admitted to Ward 30 on October 13, 1892, and examined October 14. His complaints were swelling in the legs, scrotum, abdomen, and face, and also shortness of breath, and he stated that his illness had commenced three months ago.

History:—Hereditary Tendencies.—His mother had died of some disease in which there was swelling of the legs. *Habits as to Food and Drink.*—Has always had plenty of good food, and is not a heavy drinker. *Surroundings.*—In consequence of his employment, is liable to physical strains, and when overheated is often exposed to draughts. *Previous Illnesses.*—Had measles and small-pox as a child; occasional slight rheumatic pains later.

Origin and Course of Present Illness.—About three months ago patient got a severe wetting one morning, but after drying himself at the furnace in his shop, continued to work all day. On retiring to rest that night he felt chilly, and felt also pains in his knees and ankles. Although he did not stop work, he says he has never felt well since then, and about

four weeks after he noticed his ankles swollen at night. A week ago the swelling of the ankles became suddenly much worse, and he noticed also the swelling affecting the scrotum, abdomen, and face. The quantity of urine has markedly lessened during the last week, and he has begun to suffer from shortness of breath.

State on Admission.—General nutrition and muscular development good. The only morbid appearance is the marked general dropsy.

Urinary System.—Micturition three or four times day and night; no pain. Urine—quantity (on day after admission), 23 ozs.; sp. gr., 1022; acid, no blood, albumen in small quantity, epithelial and granular casts in large quantity.

Circulatory System: Subjective Phenomena. — Since the beginning of his illness, three months ago, he has noticed palpitation on exertion, and dyspnœa; no pain. On physical examination the heart was found enlarged. The apex-beat was between the seventh and eighth ribs, $1\frac{3}{4}$ in. to left of nipple line, and there was marked epigastric pulsation. The right border of the heart at the level of the fourth costal cartilage was made out by percussion 1 in. to the right of the right border of the sternum. On auscultation, a faint systolic murmur was made out at the mitral area, and equally faint systolic and diastolic murmurs at the aortic. Accompanied by the faint diastolic murmur at the aortic area the second sound was heard accentuated. Pulse-rate, 108, regular in time and strength; its beat was forcible, but a very slight water-hammer character was perceptible. The walls of the radial artery were thickened and hard.

Respiratory System.—Respirations, 24 per min.; no pain; no cough. On physical examination the chest was found emphysematous, and impairment of the percussion note was made out at both bases posteriorly, especially on the left side. On auscultation, some weakening of the respiratory murmur,

with coarse crepitation, was found at both bases, mostly over the left.

Alimentary System.—Appetite and digestion fairly good; no thirst; bowels regular; tongue rather flabby and furred. On physical examination of the abdomen it was found to be enlarged, measuring at the level of the umbilicus 38 ins. By percussion in varying positions the presence of ascites was ascertained. The liver was found enlarged, its vertical dulness in the right nipple line amounting to $9\frac{1}{2}$ ins. The splenic dulness could not be precisely ascertained, owing to the dulness at the left base, but the organ did not appear to be enlarged.

Nervous System and *Integumentary System* are practically normal, except that for some weeks his sleep has been somewhat disturbed, and that œdema of the skin and subcutaneous tissue is markedly present.

In this case the history of a severe wetting, the occurrence of rheumatic symptoms, and the supervention after some weeks of dropsy of legs, genitals, abdomen, lung bases, and face, with diminution in the quantity of the urine, and the presence in it of albumen and casts, pointed to an acute nephritis. But we had found also affecting the patient, emphysema, a greatly dilated and hypertrophied heart, with mitral, and probably also aortic valvular disease, and atheromatous arteries. All these latter symptoms might reasonably be ascribed to the single fact of his being a blacksmith of about 40 years of age.

We remembered also that the emphysema and the cardiac and vascular conditions were certainly of some duration, and that although on inquiry we could not elicit evidence that symptoms showing weakened action of the heart had existed, we believed that there must have been some tendency to backward pressure, and that this in the case of the kidneys meant an increased liability to acute nephritic attacks.

But the question occurred to us, Had there been previously

any possibility of the existence of cirrhotic kidneys? We know that this morbid condition is often associated with the cardiac and vascular changes found in this patient, and that often, too, the renal condition is the primary one. Are we to suppose, then, that this condition of kidney has been present, and has taken its share in inducing the cardiac and vascular changes which we have found, or are we to suppose that in connexion with his circulatory system we are to recognise the foundation of the whole mischief? At the time when the patient came into hospital we were inclined to take this latter view; we felt sure that the big heart and hard arteries had been there for some considerable time, we could not say the same of the renal trouble.

But I wish now to consider with you more in detail the reasons for and against this opinion, because such a consideration will, in the first place, show you that we cannot be so absolutely sure as we might think; and, in the second place, because it may help you to form an opinion in other cases of a similar kind. When the symptoms of cirrhotic kidneys are well marked, the condition is very easily recognised; but the diagnosis is often difficult in the earlier stages, and, of course, you want to be able to diagnose it in as early a stage as possible.

From the etiological standpoint nothing definite can be elicited. He is at the age about which cirrhotic kidney becomes common; he has a history of hard work and of exposure, and of rheumatism. But all these, it may be said, apply as etiological factors as cogently to the state of his circulatory system as to his renal organs. Prolonged muscular exertion leads to dilatation and hypertrophy of the left ventricle and sclerosed arteries, as well as does contracting kidneys. It may be asked here,—To what extent can the aortic and mitral valvular disease of themselves account for the large heart? To this I would answer that, as indicated mainly by the pulse, these lesions are slight, so that for an explana-

tion of this we must look rather to increased arterial pressure.

Then as regards symptoms,—whilst dyspnœa, palpitation, and disturbed sleep are symptoms of cirrhotic Bright's disease, their occurrence in this case may well be explained in other ways. The hard pulse, so characteristic of the kidney trouble, is slightly modified in this case as the result of the valvular lesions, but still the heart-beat is strong. With the rigid artery, however, it might easily be accounted for on the theory of a primary cardiac and vascular morbid condition. Headaches, giddiness, and disturbed digestion, all of which are common symptoms of cirrhotic kidney, he has not complained of, and had he done so they also might readily be explained by the cardiac condition. The liver is markedly enlarged, evidently from congestion; and whilst the consideration of its size makes us wonder that symptoms of disturbed digestion have not been more prominent, it shows that there must have been great backward pressure, and that, therefore, the kidneys being similarly congested would be very liable to an attack of inflammation. Failing vision and the characteristic changes in the retina he presents no evidence of. In the absence, therefore, of any of the general symptoms of cirrhotic kidney, we pass now to a more detailed consideration of the urinary system itself, and to this let me now direct your attention.

We first inquired as to the quantity of urine which he had been passing previous to his illness, and he stated that he had never noticed any increase in its quantity or paleness in its appearance, and what is, as we shall see, of great consequence, that he never had to get up to micturate during the night. We also made out that he had never noticed any puffiness about his eyes in the mornings previous to his acute attack. We therefore, as already stated, were inclined to believe that his attack was one of acute inflammation, occurring in a patient with practically normal though congested kidneys

and valvular and arterial disease. But we made up our minds to watch him during the satisfactory recovery which we anticipated would occur, and to see if we could make ourselves more certain of his renal condition as time went on. We advised rest in bed, plenty of hot bottles and blankets to favour the action of the skin, a non-nitrogenous diet, attention to the action of the bowels, and a mixture of quinine and iron, with a small dose of digitalis thrice daily.

At once improvement began to show itself, with rapid increase in the quantity of urine, the dropsy disappeared, and the albumen and blood after a few days could no longer be detected. But it is to the condition of the urine that I now wish to direct your attention, and the points to which I wish specially to direct your attention are shown in the Tables.

Table I. shows the total quantity of urine passed since his admission each day, and since October 22 the proportion passed during the day and night. The day urine is the quantity passed between 8.30 A.M. and 8.30 P.M., and includes the quantity passed before the patients are supposed to be going to sleep; the night urine is the quantity passed between 8.30 P.M. and 8.30 A.M., and includes the quantity passed before the ward is cleaned in the morning.

TABLE I.

	Total.	Day.	Night.		Total.	Day.	Night.
Oct. 14,	25			Oct. 25,	66	18	48
15,	33			26,	65	21	44
16,	42			27,	61	32	29
17,	74			28,	46	24	22
18,	129			29,	69	29	40
19,	104			30,	65	21	44
20,	117			31,	41	12	29
21,	149	55	94	Nov. 1,	57	28	29
22,	110	38	72	2,	56	24	32
23,	93	26	69	3,	49	24	25
24,	80	35	45				

Table II. shows for the five days ending October 26, in

addition to the quantity of day and night urines, the specific gravity and the proportion and amount of urea. For the analysis and construction of this Table I have to thank Dr Boyd.

TABLE II.

Date.	Amount—Day.		Amount—Night.		Urea—Day Per-centage.	Urea—Night Per-centage.	Specific Gravity.		Total Urea in Grammes.	
	Ozs.	C. c.	Ozs.	C. c.			Day.	Night.	Day.	Night.
Oct.										
22	38	1078	72	2044	1·4	·9	1020	1013	15·06	18·39
23	26	738	69	1902	2·4	1·2	1030	1020	17·71	22·82
24	35	993	45	1277	1·6	1·4	1022	1020	15·88	17·87
25	18	511	48	1362	1·9	1·85	1028	1022	9·70	24·59
26	21	596	44	1249	2·4	2·85	1030	1030	14·30	35·59

From these Tables it will be seen—(1.) That the total quantity of urine passed by this patient ran up rapidly after his admission, and is now returning to what we may consider the normal amount. (2.) That the specific gravity and amount of urea are keeping high—indeed, rather above the normal. Neither of these, it is evident, indicate that cirrhotic kidneys have pre-existed or exist in this man; for, of course, were this so, the quantity of urine would, we should expect, have remained high and the specific gravity low, whilst the urea—though as regards total quantity perhaps equalling the average—would, corresponding to the increased amount of water, have been proportionately less than the average.

But notice that (3) the amount of the night urine is nearly always higher than that of the day. This is, I believe, an important point; and because, in my opinion, a sufficient amount of importance has not been directed to it in medical teaching, I propose to devote a little time to its consideration. My attention was directed to it some years ago when working as Assistant Physician in Prof. Greenfield's wards, and my friend Dr Claud Wilson, who was then Resident in

Wards 23 and 24, made some observations in connexion with it which were published in the *Lancet* of June 29, 1889.

In health the proportion of urine passed during the day hours is very much greater than that passed during the night. Roberts, for example, with a total daily quantity of 30 ozs. 6 drs. of urine, gives for the sixteen hours of waking 30 ozs. 2 drs., and for the eight hours of sleep only 4 drs. This gives as the amount of the secretion per hour, for the day a little less than 2 ozs., and for the night about $\frac{1}{2}$ dr., or splitting the total quantity into two, one representing the amount passed between 8 A.M. and 8 P.M., and the other the amount passed between 8 P.M. and 8 A.M., this would give for the day quantity about 27 ozs. and for the night about $3\frac{1}{2}$ ozs.

Remembering that the quantity of urine varies greatly in health and disease in different individuals, and also that our patient is lying in bed all day as well as all night, we cannot yet fail to be struck by the greatly altered ratio of the nocturnal to the diurnal excretion. On only one occasion is the nocturnal amount less than equal to the diurnal, in most cases it is much greater, on two occasions it is more than twice as much. Further, although the total amount of urine is diminishing, the excessive quantity of night urine still seems to hold.

Dr Claud Wilson, from his observations, came to the following conclusions as regards the proportionate quantity of diurnal and nocturnal secretion—"That in debility, however produced, the diurnal and nocturnal rates approximate to each other, that this is specially marked in cases of cardiac disease, and more so still in organic disease of the kidney. One case is especially interesting, in which the approximation between the day and night rates of excretion was sufficient to make me strongly suspect organic (waxy) disease of the kidney, though there was no noticeable polyuria nor any albumen. This patient died two months

later, when he was found to have extensive waxy disease of both kidneys as well as of other organs, although no albumen had been detected in his urine until a week before his death, and then only a trace."

With what Dr Wilson has said I can certainly agree, with one little qualification which I shall mention directly. I have known of cases where contracting kidney was diagnosed mainly by noting the altered diurnal and nocturnal excretion rate, and I have known of cases where the diagnosis was missed by failing to inquire into this. The little qualification which I referred to was as regards the altered rate in cardiac cases. I think that the cardiac cases to which Dr Wilson referred were cases like that of the patient whom we are now considering, and that in them, as in this patient, we cannot be sure that some renal mischief was not existent. Let me therefore impress upon you the importance of inquiring as to the diurnal and nocturnal excretion rate. Of course, alterations can occur in other morbid conditions, and even occasionally in health, but when it occurs habitually, and cannot be explained as due to other morbid causes, it may enable you earlier than any other symptom to contemplate the onset of organic renal disease.

To revert now to our patient W. He is going on favourably; he is now allowed a certain proportion of nitrogenous food, and in a few days he will, taking every precaution by clothing, etc., to protect him from cold, be allowed to get up and go about. We shall from time to time, however, make inquiries as regards his diurnal and nocturnal rates of urine excretion. If with a total quantity of 40 or 50 ozs. of urine we find that the nocturnal proportion shows a marked diminution, this will certainly influence us to believe that the kidneys have as yet suffered no fibroid change.

We shall also look to the specific gravity of the urine. Of course, if any such change is going on we expect that the specific gravity will diminish, but we shall note specially

if this diminishing is more marked in the case of the night urine. We shall also examine for casts. Up till now hyaline casts are still being detected from time to time.

And now, can any reason be given for the increased flow of urine during the night found in this and other such cases? I believe that it can, and to better explain it I would ask you to remember another feature of chronic Bright's disease, and that is the dropsy.

Corresponding with the large flow of urine the dropsy is slight, but what is of special importance is that it is marked about the eyes, and mostly in the mornings. That is to say, the dropsy about the eyes is occurring during the night when the patient is secreting the largest proportionate amount of urine. Further, if a patient with contracting kidneys falls asleep during the day, let us say in his chair or on the sofa, he will usually notice that his eyes are puffy when he wakes, and if you inquire you will often find evidence that during his sleep he has been secreting a considerable amount of urine.

The explanation of all this lies, I think, in the theory that along with the lowered metabolism of the night hours, or hours of sleep, there is a lessened lymphatic absorption. Without, in the light of Heidenhain's recent work,[1] accepting in its entirety the Ludwig theory, viz., that the urine secretion is in part a process of filtration and in part of osmosis, we may yet believe that between the filtered fluid in the tubules and the contents of the lymph spaces in the connective tissue of the kidney a certain amount of interchange will occur. At the time when lymphatic absorption is active this may, as Ludwig states, result in a diminution in the quantity, and possibly also in an increase in the specific gravity of the urine,—that is to say, in its concentration. When, however, the activity of lymphatic absorption is

[1] "Versuche und fragen zur Lehre von der Lymphbildung." Heidenhain, *Archiv f. d. ges. Phys.*, Bd. xlix.

lessened, a larger quantity of urine, with possibly lowered specific gravity, will flow downwards into the bladder. Similarly in the tissues generally, with lessened lymphatic absorption, these will contain a greater proportionate amount of fluid. Thus, in a locality where the tissues are lax, as in and about the eyelids, there will be an œdematous condition, which will disappear when the absorption process is more active.

Hence there will be remarked œdema about the eyes, most noticeable when the patient awakes in the morning, associated with a nocturnal flow of urine increased proportionately to the diurnal. Also a lessening of this œdema as the day wears on, associated with a diurnal flow of urine diminished proportionately to the nocturnal.

Of course, another explanation of this dropsy which has been brought forward is gravity and the position of the patient during the night.[1] Were gravity, however, the main cause, we should expect at the same time a corresponding dropsy of the ankles at night as the result of the patient walking about during the day. This, however, in cases of contracting kidneys such as we are referring to, we usually do not get.

[1] *L'albuminurie.* Lecorché et Talamon. Paris, 1888.

CASE OF PRIMARY CONTRACTING KIDNEY.

GENTLEMEN,—The case which I propose to consider with you to-day is one, I believe, of primary contracting kidney.

W. R., aged 52, a photographer, was admitted to Ward 30 on December 23, 1893, complaining of defective eyesight.

Family History.—Parents both dead, father at 42, mother older, causes of death unknown; two brothers dead, causes of death unknown; four sisters and one brother living; has had five children, all dead; two died, aged 20 and 25, from consumption.

Personal History.—Has always had a good home and been in comfortable circumstances; his work is for the most part indoors, but he gets plenty of leisure for outdoor exercise; has taken beer rather freely, but seldom drinks spirits; has had gonorrhœa, not syphilis.

Had no illness until three years ago, when he had an attack of influenza, which confined him to bed for two weeks. For a year after this he had a rather severe cough, but this was accompanied by but little expectoration, by no night-sweating, and by no loss of weight. He has always had a slight cough since this attack, but otherwise, he said, he has been as well as he was before. Six or seven weeks ago he found that his sight was failing, especially for near work, and during the past six weeks he tells us that it has gradually been becoming worse, but that some days he notices it more

than others. He also tells us that since the attack of influenza he has been troubled with headaches. These at times are very bad, especially at night, and he states that he often goes to bed feeling all right, and is awakened during the night by the headache. This headache seems to be usually occipital, but sometimes it is frontal. He distinctly states that there has been no exacerbation of the headaches during the past six weeks,—that is to say, since his eyes became affected. The headaches also never cause vomiting.

State on Admission.—He is a man of under middle height, but fairly well proportioned, and of fair muscularity. There is a slight fulness under the lower eyelids, with a semi-circular hollowing below, which seems to indicate that this fulness has at one time been more marked.

Alimentary System.—His mouth is rather dry; he is more thirsty than he used to be, and his tongue is somewhat coated. Beyond a slight tendency to constipation, there is no further abnormality. The liver and spleen are normal.

Circulatory System.—There are no subjective phenomena. Pulse is 90 per minute, regular in force and rate. The tension is high, the pulse wave is small, and the arterial walls are somewhat sclerosed and tortuous.

Heart.—Apex-beat can be felt in the fifth interspace, in the nipple line. Owing to the presence of emphysema, the percussion of the heart is a little difficult, but the left border can be made out distinctly to the left of the nipple line. On auscultation the first sound is reduplicated in all the areas, and the second sound is accentuated, and presents the "pump-valve" character in the aortic area.

Respiratory System.—He states that for the past two months he has noticed himself more short of breath than formerly. He has a slight cough with muco-purulent expectoration. His chest is distinctly emphysematous, but the physical signs of bronchitis are very slightly marked indeed; the breath sounds, somewhat feeble, all over show a

slight inspiratory harshening. There are no crepitations anywhere.

Urinary System.—He states that for the past seven weeks he has had to get up two or three times at night to pass water, and that he has been passing a considerable quantity. Since his admission the quantity has varied between 50 and 90 ozs. It is light in colour, acid in reaction, and has a specific gravity of about 1010. It also contains a small quantity of albumen, ·2 per cent.; a trace of globulin, and a few hyaline casts. The amount of urea is about 300 grns. in the twenty-four hours.

Integumentary System.—The skin is usually rather dry, and he sweats very little. There is some œdema below the lower eyelids, as stated previously, and he says that this is most noticeable in the mornings, and that it disappears as the day wears on. He did not complain of œdema of the feet, but on careful examination a little pitting over the shin bones at the ankles could be detected.

Nervous System.—Except the headache and failure of eye-sight, as previously mentioned, he has no abnormal sensations.

Examination of Eyes.—His left eye shows a scar over the lower part of the cornea. This, he says, was due to an accident with a chisel, which he sustained when he was 17 years of age. Ever since that time the sight in the left eye has been impaired, but this did not trouble him. In both eyes vision has been failing for the past six or seven weeks. He states that for about two weeks before his admission he has also noticed coloured rings around any object he looked at. Sometimes there were several rings of different colours; at other times stars appeared. He saw no flashes of light when in the dark, and he stated that he could see better by gaslight than by daylight. He also complains of severe pain above the eyes. This pain sometimes comes on suddenly, like lightning pains. These phenomena occur in connexion with both eyes, but specially with the right.

On examination the movements of the eyeballs were found unimpaired. The pupils are equal and regular in outline; they react readily and equally with accommodation, but scarcely at all to light. With the ophthalmoscope both optic discs appear blurred, but the vessels are not obscured. Numerous white patches of well-marked radiating white lines of albuminuric retinitis in both eyes. These changes, however, are more marked in the right.

Reflexes.—Superficial: Appear normal. *Deep:* Both knee and ankle jerks are well marked in both legs. No clonus can be elicited. *Organic:* All the organic reflexes are normal, with the exception of the bladder, for he tells us that sometimes he is suddenly seized with a desire to pass water, and can scarcely check it.

Cerebral and Mental Functions are normal, except that his sleep is not good, and is apt to be disturbed by dreaming.

The diagnosis which we have arrived at is that we have to deal with a case of primary contracting kidney, with the associated thickening of the arterial coats and hypertrophy and dilatation of the heart, and with its frequently concomitant emphysema. And now let us consider in detail the etiology and symptomatology of this complaint as manifested by our patient.

Etiology.—As you are all well aware, the etiology of contracting kidney is rather obscure, but foremost among the conditions which act as etiological factors are the three,—alcohol, lead poisoning, and gout. Now, in our patient you will have remarked there is no distinct history of any of these to be elicited. Of course gout is a very wide term, and in the sense in which it is used in connexion with primary contracting kidney it covers a multitude of conditions; but when we try to elicit from our patient any history of dyspepsia or such like—of symptoms which might be classed as gouty—we find ourselves baffled, for up to the attack of influenza which he suffered from three years ago, he states

that he has had no ailments whatsoever. To understand this matter better, however, let us first try to understand how such clearly-defined factors as alcohol and lead produce contracting kidney, and see if from this consideration we can get any light. One explanation of this is, that as the result of blood containing alcohol or lead circulating through the kidneys for months or years, a chronic irritation is set up, resulting in the infiltration of the interstitial tissue with leucocytes, and in the formation there of new fibrous tissue, which, contracting, leads to shrinking of the organ as a whole, with consequent pressure upon and obliteration of its tubules and glomeruli to a greater or less extent. This is chronic interstitial nephritis. Others look for an explanation of contracting kidney in "nothing but a simple degenerative atrophy of the renal parenchyma, and in a corresponding gradual increase of the interstitial connective tissue,"[1]—that is to say, they believe that, just as fibroid change takes place along certain tracts of the spinal cord when they are shut off from the influence of their trophic realm, so in the kidney when from one cause or other the nutritive power fails, a similar disappearance of the normal structural elements and an appearance of fibrous tissue will occur. Just as sclerotic changes in the spinal cord mean that the more complex and specialized elements, viz., nerve cells and nerve fibres, are from defective nutritive power no longer able to reproduce themselves in their more complex and specialized form, but disappear and are replaced by the less complex and less specialized fibrous tissue, so in the kidneys an exhausted nutritive power will mean more or less disappearance of the specialized elements, viz., the renal tubules and epithelium, and a proportionate increase in the less specialized, viz., the connective tissue. Between chronic inflammation on the one hand and degeneration on the other it is impossible to draw a line, but I think you will see that, looked at in this way,

[1] Strumpell, *Text-book of Medicine*, p. 856.

the effects will be the same. Now, it seems to me that to look upon primary contracting kidney as a degenerative rather than an inflammatory process is to take a most reasonable view.

It will explain the occurrence of the senile kidney—the contracted granular kidney which is found in old people, as being the result of slow failure of nutritive power—and it will account for the contracting kidney of the alcoholic or the lead worker, as being, as it were, a senile kidney brought on prematurely as the result of the overwork which the efforts to withstand the effect of the alcohol or lead have thrown upon it. It will also, as readily as any other theory, explain why the gouty or the rheumatic individual—or one who is dyspeptic as the result of long-continued over-eating, over-working, or mental worry—should be liable to suffer from contracting kidney. In all these we know that faulty metabolism in the organs or tissues will lead to the presence of products more or less irritating in the blood and in the kidneys, and that this, acting like alcohol or lead, will not fail to produce a premature wearing out or senility of the renal parenchyma.

Further, just as we know that the duration of life of an individual depends not only upon the wearing out to which he is subjected, but upon the amount of vitality with which he is endowed at birth, or, to be more correct, at conception, so with the kidneys. The innate vital power of these organs varies much in different individuals. Wear and tear which in one will be successfully borne for eighty or ninety years, will in another cause what may be termed senile kidneys and death at 35 or 40. Contracting kidney is markedly hereditary.

If we now apply these considerations to our patient, who at the age of 52 presents definite indications of contracting kidney, all that we can say is—(1), that from the fact that his father died at the age of 42, he may possibly, as regards

his kidneys, be hereditarily rather below par; (2), that his attack of influenza three years ago may also have aided in the using up of their nutritive energy; (3), that he may possibly have been taking too much alcohol, and that, as the result of faulty dieting, exposure, etc., some morbid material may have been circulating through his kidneys, and prematurely have used up their trophic power.

One thing seems certain, and that is, that there is no evidence of any parenchymatous nephritis, which might, as it often does, set the fibroid change in action. This is a case of *primary* contracting kidney.

Symptomatology.—Notice here that what brought our patient to the Infirmary was impaired eyesight. This exemplifies what you will often find, viz., that this disease is frequently first diagnosed by the ophthalmic surgeon. But this exemplifies also something even more important, and that is, that the disease in its earlier, and therefore more remediable, stages is to be recognised by objective signs rather than by subjective symptoms. The main explanation of this is that the impaired function of the kidneys is compensated for by extra work in other tissues and organs, notably in the circulatory system, and that, therefore, with practically entire absence of symptoms, we may have, if we remember to look for them, distinct evidence of the existence of the disease in the presence of cardiac and vascular changes. This, however, will be best understood by discussing the various systems in detail.

Circulatory System.—Here the only symptoms of derangement are that, on inquiry, we find that he has noticed himself for the past few months more easily put out of breath than formerly. He has no giddiness or other evidence of weak cerebral circulation, and no palpitation nor cardiac pain. On examination, however, we find his heart enlarged, a reduplicated first sound heard over the ventricles, and a loud "pump-valve" second sound, heard loudest in the aortic area.

The cause of the reduplicated first sound and of the pump-valve second sound I believe to be the abnormally high pressure in the left ventricle and aorta. Normally, as you know, the pressure in the left heart is supposed to be three times greater than that in the right; but in our patient, although we believe the right heart to be somewhat dilated and hypertrophied as the result of the emphysema, we believe that the left heart is proportionately still more so. The extra tension in its cavity, therefore, is the cause of the reduplication of the first sound, because it induces asynchronism in the systolic contraction, and in the sudden tension of the valve cusps and cordæ tendiniæ of the two ventricles. The extra tension is also the cause of the pump-valve aortic second sound, because of the greater pressure in and probably also dilatation of that tube. The high arterial tension is shown very well by sphygmographic tracing; and with the sphygmometer the pressure on his admission was found to be about 8.

But, now, what is the cause of the hypertrophied heart, the high tension, and the rigidity of the vessels? On this subject a great many theories have been brought forward. Johnson ascribed it to the morbid material in the blood stimulating the vasomotor nerves, and so exercising a "stop-cock" action on the small vessels, the result of which would be to induce heightened blood-pressure, hypertrophied heart, and thickened vessels. Traube's theory, as modified by Cohnheim, is that in the condition of renal inadequacy there is a hindrance to the circulation through the kidney, and that this will react and cause increased blood-pressure in the entire arterial system, and so induce hypertrophied heart, etc. But the view which I think will occur first to you is a simple one. It is, that the kidneys being inadequate, noxious materials are retained in the blood, and that thus the give-and-take interaction between blood and tissue is disturbed. The tissues will thus less readily assimilate their nutritive material, and the blood will therefore require more force to drive it on.

Thus can be explained the hypertrophy of the heart. The thickened vessels might be explained in the view that the extra pressure brought about by the hypertrophied heart entails strengthening of the arterial walls. Further, we can suppose that, just as in the heart the compensatory hypertrophy is apt to be followed in time by fibroid or fatty change in its wall, so in the vessels the hypertrophied elastic or muscular coat is apt in time to show degenerative fibroid or sclerotic changes. But note that, in whatever way induced, this hypertrophy of the heart and high arterial tension is compensatory, and consequently salutary. As cell after cell of renal epithelium is destroyed, as patch after patch of secreting tissue becomes fibrosed, and as, consequently, the renal inadequacy becomes more and more marked, all this is more and more compensated for by the forcing of an extra quantity of blood through the kidneys, and so long as the heart is equal to the extra work, and the vessels to the extra strain, no trouble is caused and no symptoms are induced. Sooner or later, however, the strain begins to wear out the machinery somewhere, but this will be better referred to later on.

Another symptom which comes for consideration under the circulatory system is the anæmia. This is a common symptom in contracting kidney, but it is not present in our patient; for in him the corpuscles were found to be close on the 5,000,000, and the hæmoglobin about 60 per cent. When we reflect that one cause of the anæmia so common in these cases is the loss of albumen by the urine, and that another is the disturbed appetite and digestion, we can understand that our patient should not present anæmia; for the amount of albumen in his urine is very small, and his appetite and digestion are fairly good.

Respiratory System.—A prominent symptom here is dyspnœa on exertion, and the usual causes of this dyspnœa are weakened action of the heart, and probably also anæmia. Among other

causes is irritability of the nerve centres; and in our patient the slight dyspnœa on exertion which he complained of may possibly be ascribed to this, for, as you have seen, his ankle and knee jerks are specially well marked. But a further cause is doubtless the emphysema, which is specially common in cases of contracting kidney, and which is markedly present in our patient. The slight cough which he occasionally suffers from will be due to the emphysema inducing from time to time attacks of bronchial catarrh.

Digestive System.—In our patient the dyspeptic symptoms, which are so often troublesome in contracting kidney, are as yet markedly absent. His tongue is perhaps slightly furred, but all he complained of was a rather dry mouth, increased thirst, and constipation, all of which can be explained by the large quantity of urine which he is passing.

Urinary System.—The urine presents, as stated, the characters of contracting kidney: it is large in quantity, pale in colour, low in specific gravity, and it presents albumen and casts. The large quantity is, of course, compensatory, and persists in these cases till a failing heart lowers the arterial tension and increases the venous. With the large quantity of urine the amount of urea per ounce is below the average, although the patient may be excreting the normal amount in the twenty-four hours. In this patient the amount of urea in the twenty-four hours is about 300 grains.

In such cases, further, the flow of urine per hour at night is specially great, hence increased frequency of micturition at night is often an important symptom. In cases of primary contracting kidney the amount of albumen is usually small, and it is important to remember that it may be absent altogether from beginning to end. In this patient it was on his admission about ·2 per cent., but it has completely disappeared since. Casts are always to be looked for, and corresponding with the atrophied and degenerated condition of the renal epithelium they are hyaline or granular in character.

Integumentary System.—His skin is dry, and he has noticed that he has not been sweating for a time. As regards dropsy, it is slight, corresponding with the large quantity of urine which he has been passing. As already stated, he has himself observed only a little puffiness of his eyes in the mornings on waking, and we on his admission observed as well a very slight tendency to pitting about his shins and ankles. An interesting question crops up here as to why in such cases the dropsy is observed only about the eyes and only in the mornings; and this latter phenomenon is all the more interesting when it is remembered that, showing itself in the mornings, it must have been occurring during the night hours when the flow of urine per hour is apt to be specially great. This point, however, I have referred to in a previous lecture.[1]

Nervous System.—As you all know, symptoms referable to the nervous system are very common in contracting kidney. This can be understood when we remember that any impurity in the blood must readily affect the sensitive nervous tissues. Hence result headaches, neuralgias, sleeplessness, Cheyne-Stokes breathing, etc. Further, we have to remember that alterations in the blood-supply—either too high pressure when the compensation is complete, or too low when it fails—will affect brain metabolism, and cause giddiness, tendency to faintness, etc. In our patient only headaches and disturbed sleep are complained of; but it is of interest to notice that his ankle and knee jerks are distinctly increased, as indicating that his nerve centres are rather more irritable than normal.

Again, you know that, as the result of the same cause, the mental functions are apt to be affected,—moroseness and irritability of temper are common associates of this condition of renal inadequacy. Our patient, happily, appears to be unaffected in this way; but one sometimes sees distinct mental impairment, and maniacal attacks supervene in such cases, specially towards the termination.

[1] "On a Case of Renal and Cardiac Disease," p. 32.

[I cannot refrain here from alluding to a patient, L., who was in Ward 30 in December last. This man had been a soldier in India, and some of you will remember him from the fact of the marks shown on his left breast, viz., two double D.'s and a B. C. Though quite quiet and orderly in the ward, he had evidently been, to use a vulgar term, a bad lot in the army; and you will remember we believed we had an explanation of this in the fact that he had had an attack of acute nephritis at the age of 17 years, before he had enlisted; and that all his symptoms evidenced the existence of secondary contracting kidney, dating from that time. The reading of his case at the clinique forcibly recalled to my mind Rudyard Kipling's story *In the Matter of a Private.* Kipling here describes how a certain private Simmons passes into an attack of male hysteria, how he shoots his comrade Losson and also his major, and how he keeps shooting right and left in the barrack square till he is at length overpowered and taken. He is, of course, hanged in hollow square of the regiment; and then Kipling tells us that the colonel says it is drink, and the chaplain says that it is the devil, but that no one thinks that it is hysteria. Well, in our patient L., I have no doubt that his misdoings in his regiment would be similarly ascribed to drink and to the devil, but I have as little doubt that they were mainly due to renal inadequacy.]

But a most important symptom which our patient presents is the albuminuric retinitis. This, or rather the disorder of vision which it entailed, was indeed what brought him to hospital. Albuminuric retinitis is said to occur in from 10 to 20 per cent. of the cases of contracting kidney, but as regards its cause we have little precise knowledge. We know that it occurs in chronic cases, and often when the general symptoms of kidney disease are slight, hence it is probably due to some morbid material in the blood. It does not seem to be so much due to the consequent cardiac and vascular changes, because we know that hypertrophied heart

and thickened vessels existing without albuminuria do not induce it. Further, in cases of albuminuric retinitis the prognosis is, as we shall see, very unfavourable, hence we may conclude that it is, at any rate, associated with some defective trophic power on the part of the nerve tissues.

Prognosis.—In contracting kidney the prognosis is always bad. The disease is progressive, but with care cases may last and work may be done often for many years. Sooner or later, however, changes occur—

1. Cardiac compensation may fail, and death occurs with symptoms of backward pressure, lung congestion, diminution of the quantity of urine, dropsy, etc.

2. Cardiac compensation remains good, but a vessel gives way in the brain, and the patient dies from cerebral hæmorrhage.

3. Pleurisy, pneumonia, peritonitis, or acute nephritis may occur.

4. Uræmia may occur.

5. The fatal issue may result from sudden œdema of the glottis or of the lung, or pulmonary apoplexy.

In our patient the favourable points are that the specific gravity of the urine is fairly good, and that the heart shows no marked sign of failing compensation, but the albuminuric retinitis makes the prognosis grave. It has been stated again and again that the duration of life in such cases is not over two years.

Treatment.—As regards diet, non-nitrogenous foods seem to be indicated in those cases, but you have to remember that if the patient is weakly, and more especially if there is any tendency to anæmia, some nitrogenous food should be allowed. The patient's feelings are often a good guide in this matter. If he feels an appetite for such, and if he can digest them, the probability is that good will result.

As regards regimen, care should be taken to keep the skin in proper working order. Flannel should be worn, and

occasional baths should be employed,—hot-water or vapour, if Turkish seem too risky. For the bowels, laxatives have usually to be employed. In addition, articles of food which have an effect in this way may be selected, such as fruit, etc. You all know the risks of constipation. Patients with contracting kidney often succumb to cerebral hæmorrhage as the result of straining in the W.C.

As regards drugs, a combination of the bromide and iodide of potassium often does good; and for the anæmia, iron, if it can be borne, may be employed. Our patient is getting the bromide and iodide, and we found his general condition and headaches relieved by occasional doses of nitro-glycerine, and by occasional hot-air baths. His headaches have also been relieved by blistering behind the ears.

A CASE OF HYDRONEPHROSIS TREATED BY ASPIRATION.

GENTLEMEN,—James W., aged 46, a shoemaker, residing in Edinburgh, was admitted to Ward 30 on January 14, 1896. His complaints were of a burning pain in the left side and across the small of the back, and he drew attention to a swelling in the left side of his abdomen.

His family history is very good, and as regards personal history, he has always been of temperate habits, and has lived in a comfortable home. When a child he had scarlet fever and small-pox. In 1868 he had an attack of what he calls dysentery. As he has never been out of the country, his diagnosis of this illness is not likely to be correct; but he seems to have been ill for several months, to have had diarrhœa and tenesmus, with pain in the left side of the abdomen, and to have passed mucus and blood. In 1879 patient attended St Mary's Hospital in London, for a complaint which he considers in every way similar to that from which he now suffers. He was treated for eight weeks, at the end of which time all his symptoms had disappeared. In 1894 he was ill for three months with what he calls "kidney trouble." He had severe back pain, swelled legs, dyspepsia, and he was very weak. He soon, however, recovered, and the only other points in his personal history are, that he has always suffered from constipation, and that when young he used to suffer considerably from having to

retain his water for a long time, owing to no convenience being at hand.

Present Illness.—On December 28 last, patient had occasion to be out of doors a good deal, and got a severe chill. He had to keep his bed during the next few days, and had fits of shivering. On the night of January 2, whilst in bed, he felt a burning pain in his left lumbar region. This pain gradually increased for a week, and then became less severe. It was at the end of this week that the patient first noticed the swelling in his abdomen. No improvement occurring, he came to the Royal Infirmary and was admitted as above. During the twelve days before his admission, patient suffered from sleeplessness; he could not lie on his right side owing to the pain, and he found relief by sitting in a doubled-up position. He told us also that for ten days previous to his admission the water which he passed was of a red colour, and in quantity much less than normal.

State on Admission.—Height, 5 ft. 6 ins.; weight, 8½ stones; muscularity, poor; face, sallow; pulse and temperature, normal.

Alimentary System.—No subjective phenomena, with the exception of constipation and occasional flatulence.

Abdomen.—On the left side of the abdomen a swelling is seen, its most prominent point being half an inch directly to the left of the umbilicus. In the nipple line the swelling extends from the costal margin above to the iliac region below. Viewed posteriorly, there is a distinct bulging on the left side. On palpation the swelling is felt to be tense and elastic, of a rounded shape, and about the size of a child's head. It can be moved slightly in the antero-posterior direction. Pressure on it at any part causes slight pain. On percussion all over it a dull note is obtained. In front the dulness extends close up to the umbilicus, so that the tumour, in growing, has pushed the descending colon towards

the right side. Otherwise the abdominal viscera are normal, as are also the circulatory, respiratory, and nervous systems.

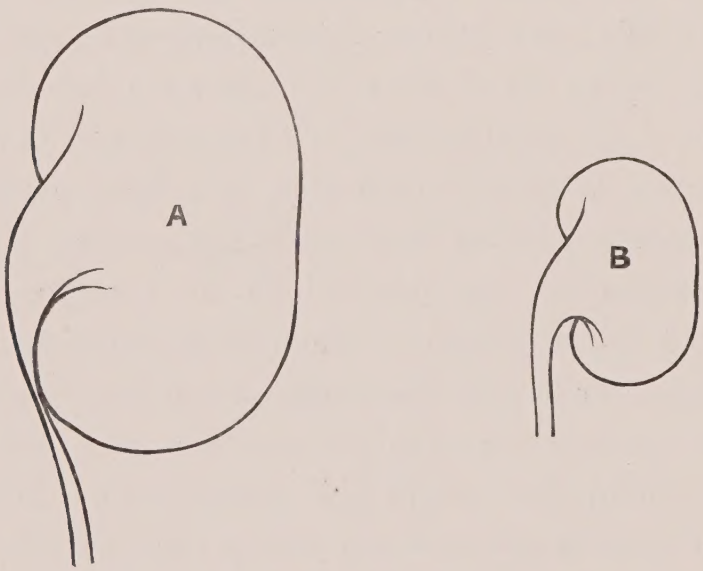
Urinary System.—Urine averages 50 ozs., pale, acid, and of a specific gravity of 1010. It contains no blood or albumen, and of urea about 5 grains per oz.

In this patient we came to the conclusion that we had a hydronephrosis of the left kidney, and believing that it was possible that it might in time empty itself along the ureter, as it had done previously in 1879, and as his symptoms presented nothing urgently requiring interference, we determined to give him some days' rest in bed, and wait to see if on this occasion also a similar fortunate development might not occur. We gave him a placebo, therefore, and watched him every day to see if we could ascertain any diminution in the size of the renal tumour, with any increase in the flow of the urine. We tried to aid this by a little gentle shampooing downwards in the direction of the ureter. No change, however, occurring, we determined to interfere, and on January 24 we aspirated. We used a small needle, and punctured rather behind the posterior axillary line, choosing this point because here the parietes are thin, and because here we should best avoid the peritoneum. During the aspiration process we kept up steady pressure all over the front of the tumour, and we drew off 22 ozs. of a fluid resembling port wine in colour, but rather turbid, and having a specific gravity of 1012. This fluid was urine—evidently dilute urine—mixed with blood, the deposit showing microscopically red blood-corpuscles, many of them more or less broken down.

After the tapping, the patient expressed himself as feeling much relieved, but it was noticed immediately that the urine was now coloured and contained blood-corpuscles. Thus we recognised that the hydronephritic sac with its bloody urine was now communicating with the bladder.

Only to a certain extent, however, for as day succeeded

day we observed that though the blood-stained urine continued, the sac was again distending. Further, with this re-accumulation in the sac, the amount of blood-staining in the urine seemed to be lessening. This we explained in the following manner:—With the sac distended as at A, the ureter was compressed, and so no escape of the sac contents occurred. With the emptying of it by aspiration, the ureter became patent as at B, and so the urine contained blood, etc. But the fluid collected again, and the sac again began to compress the ureter, until a condition much like that of the original one had been established.



On February 8 the tumour appeared to be about as large as it had been before, and although we realized from the condition of the urine that the sac was still discharging into the bladder to some extent, we judged it best to aspirate again. This we did on February 9, and evacuated 26½ ozs. of fluid, of sp. gr. 1002, and containing blood and urea as before, but rather lighter in colour. Since then there has been practically no further accumulation, and the urine is perfectly clear. In amount it is about 50 ozs., and in sp. gr. 1015. We therefore believe that the sac is now collapsed, and that although this kidney is, owing to the effects of the hydro-

nephrosis, probably to a large extent destroyed, any urine which it is secreting is passing readily down to the bladder.

But, now, what is the cause of the hydronephrosis in this patient? The commonest cause in such instances is calculus blocking up the ureter; and notice, that to produce a hydronephrosis the ureter block must not be complete. Sudden complete obstruction by a calculus leads to obstruction behind and rapid dilatation, but this is followed by subsequent atrophy of the kidney. It is only when the obstruction is incomplete that the kidney can go on secreting sufficiently to produce sacculation, so that if there is a calculus in this case, it has not been producing complete obstruction. Other causes are, narrowing of the ureter, by tumours or cicatricial contraction after inflammation.

In our patient there is no history pointing to renal calculus, and on the two occasions on which we punctured the sac we watched carefully to find if the needle grated against anything like a calculus, but it did not do so. After each tapping we also carefully palpated to see if a stone could be felt, but without success. Then in our patient there is no evidence of tumour, pelvic or abdominal, pressing on the ureter. A point of some importance, however, is that in 1868 he is said to have had an attack of dysentery. During this attack he seems to have been seriously ill, and to have passed mucus and blood in considerable quantity, and to have had straining. As dysentery is rare in this country, we think it possible that there may have been some enteritis and consequent ulceration about the colon or sigmoid flexure, and we believe it possible that the cicatricial contraction after this may have to some extent constricted the left ureter. What had been the cause of the aggravation of the resulting dilatation in 1879, and again in January last, it is difficult to say. We can easily understand, however, that with a ureter considerably obstructed either from calculus or constriction, some practically accidental cause,

such as movement or twisting of the body, may have produced aggravation of the obstruction. Then, with the sac being distended more and more, bleeding was likely to have occurred, so that on aspiration we found urine mixed with blood.

The effects of treatment in this case have been very satisfactory. Any obstruction that there may be in connexion with the left ureter is not now causing dilatation, and this affected kidney, and the right kidney which is probably hypertrophied, are together efficiently discharging urea and other urinary products. He was discharged on March 11, 1896, feeling quite well and ready for work; and palpation of the left lumbar region revealed neither tenderness nor swelling. We have advised him to wear an abdominal bandage, and we trust that with care to avoid sudden movement or twisting, and care also as regards cold, he may keep fairly well.

ON DIABETES MELLITUS.

GENTLEMEN,—We have had in our Ward lately a number of cases of diabetes mellitus, distinctly greater than usual, and we find on inquiry that a similar experience has fallen to the physicians in other parts of our Infirmary. On inquiring of Dr Gillespie, our Medical Registrar, I found that for the last seven years the numbers of cases treated for this disease in the hospital have been as follows:—1888–89, 16; 1889–90, 17; 1890–91, 23; 1891–92, 27; 1892–93, 32; 1893–94, 42; 1894–95, 30. It will therefore be seen that there has been a very considerable increase in the number of cases admitted during the last three years. Dr Gillespie has further pointed out to me that there were three periods during which these cases were specially numerous, viz., October and November 1891, December and January 1892–93, and February and March 1894. It is noteworthy that each of these periods coincides with or follows periods during which epidemic influenza was noted to be specially prevalent. Remembering the depressing effect of influenza on the bodily nutrition, we are not surprised at such results. Doubtless influenza has left other affections in its train, and doubtless also certain of our cases of diabetes mellitus can be shown to be ascribable to other etiological factors, but the special frequence of diabetes during late years, and its association, as regards time, with influenza, are well worthy of remark.

The case of diabetes mellitus which I propose to discuss

with you to-day is that of a man, T. L., æt. 50, a labourer, who was admitted to the Infirmary on 31st October 1895. His complaints were that he was always hungry and very thirsty, and that he had to make water with great frequency.

His family history is fairly satisfactory, showing no tendency to diabetes nor to any other constitutional complaint. He has had the ordinary illnesses of childhood, and has had measles twice, the second attack being when he was 20 years of age. When 27 years of age he had an attack of gonorrhœa, and a year later, at which time he was serving as a soldier in India, he had an attack of jungle fever. For the last fifteen years he has been working as a labourer in Leith. He describes himself as having been a moderate drinker, and he states that his surroundings at home and at work have been fairly comfortable.

His present illness seems to have begun about twelv e months ago. After what was apparently a slight attack of influenza, he remarked a gradual increase of thirst and appetite, and increasing frequency of micturition. He tells us that about every twenty minutes he had to leave his work for the double purpose of obtaining drink and making water. He noticed that he was especially thirsty after meals, and says that it was no uncommon thing for him to drink as much as a quart of water after breakfast. If he had of necessity to do without drink, his mouth had a sticky sensation and his tongue grew hot and dry. During the course of his illness he has been habitually constipated, the bowels being moved at intervals of three or four days. He has also noticed that he has been rapidly losing weight. Twelve months ago he weighed 13 stones 2 lbs., three months ago his weight was 10 stones 12 lbs., at present it is only 10 stones $1\frac{1}{4}$ lbs. Although not compelled to give up his work, he has been feeling himself grow weaker, and so has sought admission to the Infirmary.

Present Condition.—Patient is 5 feet 8 inches in height,

and his weight, as stated, is 10 stones $1\frac{1}{4}$ lbs. He has the appearance of having been a well-developed man, but his muscles are now comparatively small and flabby.

Alimentary System.—Patient has lost six teeth; those remaining are nearly all more or less carious, and he states that he has noticed that his teeth have been decaying more rapidly during the past twelve months. His tongue is red and dry, and somewhat furred posteriorly. The oral secretion gives a markedly acid reaction with test paper. His appetite is voracious and his thirst insatiable. His digestive power is very good, and he volunteered the information that previous to his illness he could not have eaten or digested what he can do now. Bowels are somewhat constipated. On physical examination of the digestive organs the only noteworthy point is that the liver is slightly enlarged. Its percussion dulness in the nipple line is over 8 inches, and it can be distinctly recognised by palpation below the inferior costal margin.

Hæmopoietic System.—No enlargement of the lymphatic glands or of the spleen can be detected.

Circulatory System.—He has no subjective phenomena, except that he feels more easily tired and more easily put out of breath than formerly. Examination of the blood shows its specific gravity to be 1056 (Roy's method), with 6,100,000 red corpuscles, and 106 per cent. of hæmoglobin. The heart is of normal size, and shows no evidence of valvular disease. His pulse is 70 per minute, regular, soft, and compressible. The radial artery is somewhat tortuous, and its wall slightly thickened.

Respiratory System.—He has no subjective phenomena, and careful examination of the lungs fails to reveal anything abnormal.

Integumentary System.—His skin is harsh and dry. There are no eruptions, but over the metatarso-pharyngeal joint of each thumb there are slight scabs. He states that these

are the results of a slight injury inflicted some weeks ago, and he states that he has noticed that slight injuries to his hands take a longer time to heal now than they did formerly. There is no apparent œdema, but if we press firmly with the point of the finger on the shin bone near the ankle, we find a very slight amount of pitting. This indicates that in spite of his polyuria there is a slight tendency for the transudation of serum into his tissues at the dependent parts.

Urinary System.—The patient has balanitis and some pain and irritation on micturition. This began twelve months ago with his present illness. There is increased frequency of micturition, both night and day. Urine, amount 250 ozs., pale and slightly opaque. It has an odour somewhat like that of sour milk, its reaction is acid, and specific gravity 1032. It contains sugar to the extent of about 8000 grains per day. The addition of caustic potass in the first stage of Moore's test causes a cloud, showing that the phosphates are abundant. There is no albumen or other abnormal constituent.

Nervous System.—His cerebral and mental functions are normal, but he states that he has noticed that his eyesight has been failing for the last three years, and specially so during the last year. The cutaneous reflexes — plantar, cremasteric, and abdominal—are very well marked. The deep reflexes, knee and ankle, are not present. There is no impairment of motion or sensation. His temperature is as a rule subnormal. Such being the phenomena which our patient presents, let us now consider his case in a little more detail.

Etiology.—Diabetes mellitus is, as you know, more frequent in men than in women, and our patient is rather above the age at which the disease is most prone to show itself. In our patient there is nothing in the family history indicating diabetes, or any other debilitating condition. This, however,

is not surprising. Although diabetes is markedly hereditary, he is beyond the age at which heredity plays its most important *rôle.* Thus, for example, of the last twelve cases of diabetes in my ward, only two showed heredity as regards this disease, and these were aged respectively 15 and 17 years, the average age of the others being over 40 years. Excluding heredity, therefore, we must look for something in his personal history which has so lowered his nutrition as to allow the diabetes to supervene. Here the points that call for remark are—that first as a soldier, and later as a labourer, he must have gone through a great deal of wearing-out work, and have suffered many exposures. Probably also he has taken more alcohol than he believes he has. There is no doubt that in a very large proportion of our Infirmary cases of diabetes an alcoholic history is obtainable, and we know that alcohol, like morphia or chloroform, is very apt to produce glycosuria. We know that drinking bouts are often accompanied by temporary glycosuria, and if drinking bouts become common, we can easily understand that the glycosuria may become permanent. Further, you will remember that in another case of diabetes which we had in our wards lately, the symptoms of diabetes were distinctly noticed to have followed the last of a series of drinking bouts.

Hard work, exposure, and alcohol have all therefore had their share in inducing his disease. But I think we are right in saying that the attack of influenza which he suffered from about a year ago has been, as it were, the "last straw," and that it has precipitated the onset of the diabetes.

Symptoms.—A good general idea of the symptoms of diabetes mellitus can be obtained from the analogy of the process of osmosis. Thus the blood, owing to the large amount of glucose in it, will be continuously drawing in to itself fluid from the alimentary canal and from the tissues, and in this way may be explained the thirst, the dryness of the mouth and of the tissues generally, and the usually

present constipation. Then the glucose, with the fluid which it draws to itself in the blood, can filter readily, and hence may be explained the polyuria and glycosuria. Further, as fast as the glucose is being eliminated by the kidneys, more of it is being sent into the blood, and as fast as fluid is being absorbed from the alimentary canal and tissues, more of it is being ingested by drinking, and so the process goes on. All the symptoms of diabetes cannot be explained in this mechanical way, but many of them can. This we shall understand best by considering them in detail as they occur in connexion with the various systems.

Digestive System.—The dryness of the mouth and thirst need no further reference; but we find in our patient, as we invariably find in diabetes, that the secretions of the mouth are markedly acid. It has been said that in diabetes the saliva is acid, but this is not so. In this patient, as in all our other cases, we have found that if we produce a copious flow of saliva by stimulating the tip of the tongue with the faradic current, the reaction of the pure fluid is alkaline. The acid reaction of the secretion in the mouth, therefore, is not due to acid saliva, but probably to some acid decomposition. To this acidity is ascribed the rapid caries of the teeth noticed in this disease. In diabetes the appetite is voracious, a symptom which we can readily understand when we remember the enormous loss of oxidizable material which the glycosuria entails. Associated with this, the digestive power is specially good. Our patient tells us that he can now eat and digest quantities of food in a way which he could not have done previous to the onset of his illness. Further, we have had patients who, previous to their diabetes, suffered from dyspepsia even though they attended carefully to their diet, and who, after the disease had supervened, could eat and digest, as they expressed it, anything. In this we can recognise one of the many examples of the "*vis medicatrix naturæ*"; the great loss of sugar

rendering the ingestion of more food necessary, Nature improves the appetite and digestive power accordingly. The slight enlargement of the liver which this patient presents is common in diabetes, and is probably due simply to hyperæmia.

Hæmopoietic System.—This is apparently normal; we find no enlargement of lymphatic glands or of the spleen.

Circulatory System.—In most cases of diabetes, as in our patient, examination of the heart and bloodvessels shows them to be fairly healthy. The pulse may be slightly quickened and slightly more compressible than in health, explicable on the theory that the heart as well as the other tissues of the body suffers from a lowered nutritive power. But in connexion with the circulatory system in diabetes, the condition of the blood is specially important. In our patient the number of red corpuscles is 6,100,000, and the hæmoglobin is 106 per cent. Now, this extra richness in corpuscles and hæmoglobin which is frequent in this disease is explained by many as being due to a concentration or a thickening of the blood, the result of the polyuria. But were this the case, the specific gravity of the blood would be correspondingly increased, and, further, we should find that the specific gravity, the number of corpuscles, and the percentage of hæmoglobin, should vary greatly with the amount of food and specially of water ingested. On this subject we have been making some observations during the session. I need not give you the results in detail, but I shall tell you what we have found in this patient (L.), and in the other diabetic (A.) whom we have at present in the ward. This will be sufficient for my purpose.

In these men we have examined the blood on several occasions between 5 and 6 A.M., and again between 10 and 11 A.M. At the earlier period they had had no food nor water for several hours; at the latter they had had breakfast and had drunk copiously of water.

The following gives an idea of the results :—

Between 5 and 6 A.M.

(L.)	Corpuscles 6,232,000.	Hæmoglobin	110 per cent.	Sp. gr.	1058.
(A.)	„ 6,316,000.	„	100 „	„	1053.

Between 10 and 11 A.M.

(L.)	Corpuscles 6,100,000.	Hæmoglobin	106 per cent.	Sp. gr.	1055.
(A.)	„ 5,056,000.	„	100 „	„	1050.

These results show that after ingestion of food and a large quantity of water, a slight diminution alike in the specific gravity, corpuscles, and hæmoglobin is remarked, but the fact which stands out prominently is that without any marked alteration from the normal in the specific gravity of the blood the proportion of corpuscles and of hæmoglobin remains specially high. I am therefore in the habit of ascribing this characteristic of the blood in diabetes again to the "*vis medicatrix naturæ*." The amount of oxidizable material being lessened by the enormous loss of sugar in the urine, Nature tends to compensate for this by increasing the oxidizing elements in the blood, viz., the corpuscles and hæmoglobin. Something analogous seems to occur in other conditions, notably in starvation. If an animal be completely deprived of food, the nutrition of the more important tissues of the nervous system and the heart is obtained by the using up of the less important, *e.g.*, the fat, muscles, glands, etc.; and with a rapid diminution of the body-weight as a whole, the blood seems to show little diminution either as regards corpuscles or hæmoglobin.

It must not be forgotten that in certain cases of diabetes a lessening in the richness of the blood will be found. I think, however, I am right in saying that in the earlier cases, and in cases which have still some vigour left, it will be found the rule to meet with no diminution, but even a distinct increase in this respect. Such at least has been my experience; and when I recall to your minds the pink cheeks and

red lips which even the emaciated of the diabetics lately in our wards have presented, you will, I think, agree with me in regarding anæmia as anything but a common associate of this disease.

Respiratory System.—This in our patient is practically normal. As you all know, lung phthisis and inflammatory affections ending occasionally in lung gangrene are regarded as being of no infrequent occurrence in diabetes, and have been ascribed either specially to some disordered condition of the trophic nerves of the pulmonary organs, viz., the vagi, or to a general lowering of nutrition. This association is shown in the table at page 76; here all that need be said is that out of 50 fatal cases of diabetes, 16 died of phthisis and 6 of pneumonia or lung gangrene. But this is not all, for of the 24 cases out of the 50 which died of coma, in at least 8 was there distinct evidence of the existence of chronic or acute lung disease.

Integumentary System.—Our patient does not complain of itching of the skin, nor are there any boils or carbuncles. But the skin feels harsh and dry, and in the scabs over his knuckles we see indications that skin wounds or abrasions are taking a longer time to heal now than they did formerly. This we shall refer to more fully when we consider the trophic condition of the tissues in diabetes.

Urinary System.—The balanitis present in this case is due to the sugar in the urine irritating the mucous membrane over which it flows, and is a common feature in diabetes. The increased frequency of micturition, whilst probably also in part due to this irritation, is mainly, of course, the result of the large quantity of water secreted, some 250 ozs. This, supposing that the ordinary capacity of his bladder is 12 or 14 ozs., means that it must be emptied some eighteen or twenty times in the twenty-four hours. Usually in diabetes, as in health, the amount of urine passed per hour is distinctly greater during the day hours than during the night hours,

this being accounted for by the food and water ingested during the day period. With this we usually find that the specific gravity of the urine and the amount of glucose and urea excreted is greater during the day hours. But it is not to be forgotten that the proportion per hour of day urine and night urine is markedly altered from that of health. In a certain number of our cases of diabetes the proportion of urine passed per hour during the night has been as great as that passed during the day, in some indeed distinctly greater, resembling in this respect what we so often find in the polyuria of contracting kidney. Further, in such instances the differences between night and day urine as regards specific gravity and amount of sugar and urea we have again and again found to be not very great, all of which observations seem to indicate that in the production of the polyuria and glycosuria of diabetes mellitus other factors than those of simple osmosis must be at work.

In our patient the following represents the state of the urine before and after the administration of diabetic diet:—

1. Average for six days on ordinary diet.

Quantity.	Specific Gravity.	Sugar.	Urea.
256 ozs.	1040.	8154 grs.	1101 grs.
highest, 292 ozs.	highest, 1044.	highest, 13,206 grs.	highest, 1916 grs.
lowest, 204 ozs.	lowest, 1032.	lowest, 5313 grs.	lowest, 549 grs.

2. Average for six days on diabetic diet.

Quantity.	Specific Gravity.	Sugar.	Urea.
96 ozs.	1038.	1886 grs.	1170 grs.
highest, 120 ozs.	highest, 1043.	highest, 2316 grs.	highest, 1400 grs.
lowest, 72 ozs.	lowest, 1032.	lowest, 1680 grs.	lowest, 756 grs.

Here it will be noticed that the main difference in the urine before and after the diet is in its quantity and in the amount of sugar. Less glucose passing into the blood from the food means less to be excreted by the urine, and means

also lessened osmosis, so that there will be less thirst and less urine passed. But the large amount of nitrogenous food explains why the amount of urea should not be diminished after the administration of diabetic diet, and this and the lessened amount of water in the urine accounts for the persistence of the high specific gravity. We have found also, as stated previously, that in our patient the phosphates seem increased, and this both before and after the diet was employed. We have tested for acetone with Lugol's test —a few drops of nitroprusside of sodium and caustic soda —but the red colour did not give place to the purple on the addition of acetic acid.

Reproductive System.—The lessening of the sexual desire which is common in diabetes does not seem to have occurred in this patient, and in this connexion it is interesting to note also that his cremaster reflex is specially well marked.

Nervous System.—Our patient is by no means dull or apathetic. His superficial reflexes—plantar, cremasteric, abdominal, and epigastric—are all well marked; but his deep reflexes—*i.e.*, the knee and ankle jerks—are absent. This presence of, or perhaps increase in, the superficial reflexes, and lessening of the deep, we have found to be very common in our diabetic cases. There is no evidence of peripheral neuritis. On looking up my notes, I find that of the 15 cases of diabetes which we have had in our wards lately, only one had peripheral neuritis. This affected only the left leg, and you will remember that we explained this limitation as being probably the result of an old injury sustained by that limb.

Our patient's sight is fairly good, his accommodation is very good for his age, and there is no evidence whatever of cataract. Here you may ask, What are the causes of the diabetic cataract and of the neuritis? Both of these have been ascribed directly to the sugar in the blood,—the cataract

to its causing drying of the lens by osmosis, the neuritis to its giving rise to the development in the blood or tissues of toxic substances, acet-acetic acid or crotonic acid, or whatever they may be, and thus producing neuritis, just as alcohol or lead or other toxic substances can produce it. Probably, however, the causation of these is not so simple, and this leads us to the consideration of the trophic condition in diabetes.

Trophic Condition.—That there is great disturbance here is evidenced by the rapid loss of weight. This is associated with the drain upon the body from the glycosuria and increased excretion of urea. The impairment in this trophic power is also indicated in our patient by the fact that wounds or abrasions of the skin of the hands have been noticed to take a much longer time to heal than they did formerly. In this connexion we had two cases in our wards lately, which showed this impaired nutritive condition to a much greater extent than this patient does. One of these was a patient, M., who was sent to us from the surgical house. He had gone there to be treated for deep-seated inflammation and suppuration in the palm of his left hand, which he believed had been due to a poisoned wound. The effects of treatment in the surgical house being most unsatisfactory, his urine was tested, and found to contain sugar. When he came down to us in Ward 30 we had little difficulty in making out that the diabetes had been existent for months, although unknown to himself, and that the inflammatory changes and absence of healing power in his hand were to be ascribed to this disease rather than to anything of the nature of a poisonous substance having been allowed to enter his tissues. The second case occurred in my ward two years ago, and illustrates even better than this how diabetes mellitus may reveal its presence by trophic disturbance. A man, R., aged 30, had been knocked over in the street by a van, and one of the wheels either ran

over or bruised his right thigh. He was brought at once from the street where the accident had occurred into the Infirmary to the surgical wards. There it was found that no fracture had occurred, and the injury appeared to be simply a bruise, which it was expected would be recovered from in a comparatively short time. After a few days, however, it was noticed that inflammatory and suppurative changes were occurring at the seat of the injury. Incisions were made into these, but the evacuations of pus brought about no improvement. In the course of a few days inflammatory and suppurative changes were noticed to be extending up and down the thigh, the pus burrowing between the muscles and in the subcutaneous tissue, so that the skin over the greater part of the anterior and inner aspect of the thigh sloughed. The urine was then examined, and found to contain sugar, and he was sent down to our ward, where after about two weeks he died. On careful inquiry we found that this man had noticed increased urination and increased thirst for some six months before his accident, and taking everything into consideration, we came to the conclusion that in causing this patient's death the diabetes was the more important factor. Lastly, in connexion with the trophic condition, we have to remember the liability of diabetics to such diseases as phthisis, pneumonia, lung gangrene, and erysipelas.

Pathology of Diabetes.—In a clinical lecture, a detailed consideration of the pathology would be inappropriate, but I think that some good general ideas on this subject can be brought to your minds by regarding the pathological condition as being to a large extent the result of one or other or all of these three main factors:—

1. That the starchy and saccharine constituents of the food, transformed by the digestive process into grape sugar, are carried by the portal system to the liver. There, instead of being transformed into glycogen and stored up,

this grape sugar is allowed to pass unchanged by the hepatic vein and inferior cava to the heart and arteries.

2. That the starchy and saccharine constituents of the food, transformed by the digestive act into grape sugar, are carried by the portal system to the liver. There this grape sugar is transformed into glycogen, but this glycogen is retransformed into grape sugar in more than normal rapidity and degree, and sent as such by the hepatic vein and vena cava to the heart and arteries.

3. That the starchy and saccharine constituents of the food, transformed by the digestive process into grape sugar, are carried by the portal system to the liver and transformed into glycogen. This glycogen is retransformed into grape sugar in normal degree, and sent in normal amounts by the hepatic vein and vena cava to the heart and arteries. But the tissues, owing to a general defect in their nutritive powers, are unable to assimilate this grape sugar, and so it accumulates in the blood and produces the symptoms of diabetes.

Now it must not for a moment be supposed that this comprises all the pathological factors of the disease. Glycosuria can occur on a flesh diet, and can be induced artificially in animals when completely deprived of food, thus showing that grape sugar can be formed from flesh food and from proteid tissue metabolism. But if you ask me to which of the three factors I ascribe the most importance, I unhesitatingly say, to the third. It seems to me that diabetes is a disease not so much the result of disordered function of one organ, the liver, as of some disordered trophic condition, affecting the tissues generally. I am aware that destruction of one organ, the pancreas, can produce glycosuria; I am aware that in this case the onset of the disease can be explained on the theory that the resulting cessation of the internal secretion of the pancreas renders the tissues unable to assimilate the sugar brought to them by the blood; and

I am aware that a like cessation of some internal secretion of the liver may have a similar effect. But I think that a study of the etiology of the disease, its marked heredity, and its tendency to occur when, as the result of some innate or acquired conditions, there is impairment of the nutritive power as a whole, all point to some more general pathological cause.

Prognosis.—In our patient the immediate prognosis is fairly favourable, but the ultimate prognosis is bad. His age and good general condition are in his favour, but the loss of sugar and urea is great, and the effects of diet are not proving of any great avail, so that we cannot but prognosticate unfavourably for him in no long time, probably within the next two years. How is his case likely to terminate? To form an idea on this point I give you a summary of the causes of death in 50 cases of diabetes as detailed in the pathological records of the Edinburgh Royal Infirmary, and from these you will, I think, be able to draw your own conclusions.

No. of Cases.	Cause of Death.	Males.	Females.	Average Age of Males.	Average Age of Females.
24	Coma.	17	7	31	25
16	Phthisis.	12	4	31	24
6	Pneumonia or gangrene of lung.	All	...	36	...
4	Erysipelas or gangrene of external parts, the result of accident or operation.	3	1	...	...

This shows that coma is the commonest cause of death, and that next follows phthisis, the ages being in each case much the same. Pneumonia and gangrene of the lung are less frequent causes, and tend to occur at a rather later age. Other causes of death are what might be called accidental.

I cannot refrain at this stage from mentioning that in

the pathological reports of the Infirmary for the year 1851 I find that in two cases of diabetes which had died of coma, atrophy of the pancreas is noted to have been found by the then pathologist, Dr W. T. Gairdner. This is, of course, interesting in connexion with the more recent investigations as regards pancreatic diabetes.

Treatment.—Our patient has been put upon diabetic diet, and we are doing what we can to keep his skin function in good order. Warm flannel clothing is of course essential for this, but hot baths, or even for patients who can stand it, Russian or Turkish baths at intervals, may be employed. As regards drugs, with the exception of opium or codeia, we do not know of any which have a marked effect on the glycosuria, but we know that with such treatment as cod-liver oil, and with arsenic in increasing doses, we often get general improvement, and these drugs we are now giving to our patient. We have seen cases which did remarkably well on milk alone, and I had a patient lately who seemed to be improved by euonymin, which I gave him on the theory that whatever affected beneficially the biliary function of the liver might have a like effect on the glycogenic. Then we must remember that whatever improves the mental and general condition has a beneficial effect on the disease, and so we must endeavour to arrange that diabetic patients be shielded from all business cares and worry, and that they should have all the good which change of air and scene alone can give.

If in time the patient declares that he can no longer eat and digest the gluten bread and almond cakes, or if the craving for wheaten bread and potatoes can be no longer resisted, the best form in which to give these is toasted. In such circumstances we not infrequently find that the allowance of a little toasted bread or chip potatoes may appear to be rather beneficial. In time, too, opium or some of its derivatives may be required, but these should only be

used with caution. For the coma, when it occurs, little can be done. On the theory that it is due to the liberation of some acid, the injection into a vein, or subcutaneously, of a 3 per cent. solution of sodium bicarbonate has been advised. So also has the inhalation of oxygen, but neither of these has been found to be of much use.

TWO CASES OF LIVER CIRRHOSIS IN CHILDREN.

GENTLEMEN,—Cases of cirrhosis of the liver in children are distinctly uncommon. Thus, whilst in the post-mortem records of the Royal Infirmary I find in 710 adult cases there were eight of liver cirrhosis, in the same records of the Sick Children's Hospital I find that in the same number of cases there is not one of this disease. Yet within the last year we have had in the ward two examples of this affection. In both of these, as we shall see, there were indications of tubercular mischief, and inasmuch as in other cases of this disease in children which have been reported there have frequently occurred manifestations of tubercle, and as in children the disease cirrhosis presents certain important peculiarities, I have thought that we might, with profit, spend our time to-day in the consideration of those cases.

The first is that of a little boy, James C., aged 6, from Cowdenbeath, who was admitted to Ward 30 on March 16, 1895. His complaints on admission were swelling of and pain in the abdomen, and occasional diarrhœa, and it was stated that he had been ill for about two years. His family history was good; his father and mother are both healthy looking. There are three other members of the family, all of whom are in good health, and no family history of tubercular disease can be elicited.

Personal History.—He was not weaned until after twelve months old, and from this period onwards he has been in the habit of taking much the same kind of food as his parents. Up to the time of his being taken ill the house in which he lived was dry and healthy.

Present Illness.—This dates from an attack of measles two years before his admission (1893). This left the patient decidedly weaker, and about eighteen months before admission swelling of the abdomen was first noted. This swelling became more and more marked, but no other symptom seems to have appeared until New Year 1895, when diarrhœa set in. The stools were noticed to be greenish in colour, and had a fœtid odour. About this time, also, slight pains, referred to the epigastrium, but not particularly associated with the ingestion of food, were complained of. The pains got worse as time went on, till at last the patient was caused to cry out with them, and was kept awake at night. As he continued to get worse, he was recommended to come to the Infirmary, and was admitted as above.

State on Admission.—He is fairly well grown for his age, but is lacking in muscularity, and his thin arms and legs contrast markedly with the large abdomen. The conjunctivæ show a distinctly yellow tinge. His temperature is normal; his pulse is usually about 90. He presents at present none of the appearances of suffering; indeed, he looks bright, contented, and happy.

Alimentary System.—The tongue is clean and pink in colour. He is always keen for his food; in fact, he has no sooner finished one meal than he wants to know when he is going to get the next. He has occasionally some pain in the epigastric region, but this pain bears no relation to the taking of food. He has no vomiting, and at present the bowels are regular. The fæces are fairly well coloured.

On examination, the abdomen is large and globular in shape. Branches of the internal mammary and epigastric

veins are seen to be distended, and to be coursing upwards over the abdominal wall. At times a transverse ridging (Wyllie's "ladder pattern"), due to coils of rather distended intestine, can be made out on the thinned abdominal wall, and slight peristaltic movements occasionally show.

On palpation the abdomen is found to be soft and elastic, and no pain can be elicited anywhere, even on firm pressure. The enlarged liver can easily be detected by palpation. Its lower border is felt rather sharp and hard, but here and there over the edge and upon the anterior surface of the organ distinct nodulation is to be felt. By percussion, deep liver dulness is found to begin in the nipple line, about the third rib, and to extend one inch below the costal margin. In the nipple line, therefore, the extent of liver dulness is about $5\frac{1}{2}$ inches. In the middle line the lower border of the liver extends 2 inches below the lower border of the xiphisternum. The spleen is markedly enlarged. It can be palpated in the left hypochondriac and upper lumbar regions. On percussion, its extent in its long diameter is found to be over $6\frac{1}{2}$ inches.

Hæmopoietic System.—No distinctly enlarged lymphatic glands can be felt in the mesentery or elsewhere.

Circulatory System.—This is practically normal. The pulse, however, is small, and of rather low tension.

Respiratory System.—Practically normal.

Urinary System.—No subjective phenomena. The urine is dark amber in colour, acid in reaction, and sp. gr. 1022. Except a trace of bile, it contains no abnormal constituents.

In the ward this boy was dieted at first on milk and starchy food; afterwards he got a little fish, chicken, and pudding. As to drugs, we gave him ℥5 liq. hydrarg. perchlor. with ℥3 liq. morphiæ for the first two weeks. After this he got simply the liq. hydrarg. We also, during the last few weeks he was in hospital, rubbed in little pieces, the size of a pea, of the unguentum hydrarg. every night over the abdomen. During his stay in hospital he was bright and

happy, and free from pain; but in his condition we remarked no distinct improvement. His weight remained very much as it had been on admission, *i.e.*, 3 st. 3 lbs. His abdomen remained large, and there was no alteration in the size of the liver and spleen. He left the hospital on July 16, and the doctor who was in attendance now informs me that since his return home he got gradually weaker, that ascites supervened, and that he died in the end of last December.

The next case is that of the patient I show you, Bridget G., aged 10, a schoolgirl, residing in Edinburgh, and who was admitted 18th January 1896, complaining of swelling of the abdomen, occasional pains in the right side, and troublesome cough and spit.

Family History.—Her father died of heart disease; her mother is alive and healthy. She has two sisters and two brothers alive and well. One brother died after vaccination, and there was one child still-born.

Personal History.—Although her home is fairly comfortable, her food has all her life been not very good. She has had none of the ordinary diseases of children, but her mother states that she has had a cough practically all her life.

Present Illness.—According to her mother's statement she has been delicate ever since a fall down the stairs which she had when she was two years of age. This accident seems to have weakened her, and the next important point is that about a year after this, when she was three years of age, she awoke one night with severe pain in the right side. Hot fomentations were applied, and the pain subsided, but in the morning it was noticed that she was jaundiced. This jaundice does not seem to have been very bad, for, on her being taken next morning to the Children's Hospital, she was treated there as an out-patient. Although apparently not seriously ill, she has remained weakly; she has had cough in the winter months, and frequent spitting of blood. Two years ago, as

she was feeble, and as the jaundice seemed rather worse, she was brought to the Royal Infirmary, and admitted to Ward 24. After staying some weeks there, she was discharged improved, but she was still weak, and the slight jaundice had never completely gone. Last winter her cough was specially severe, and she brought up larger than usual quantities of blood. It was then that the enlargement of the abdomen seems to have been remarked; her mother states that her clothes were tight one day and loose the next. She was treated at the Dispensary, and with the advent of summer she improved, and was able to go to school. As the winter came on she got ill again; she complained of occasional pain in the right side. Her cough became worse, and so she was brought to the Infirmary.

State on Admission.—She is a dark-haired, dark-eyed, rather gipsy-looking girl. She is thin and anæmic, and her skin is dry and earthy-looking. There is slight yellow coloration of the conjunctiva. The pulse and temperature are practically normal.

Alimentary System.—Her appetite is variable; at times she can eat great quantities, at other times very little. The tongue is clean, and the lips and gums are well coloured. She complains of no discomfort after eating. Bowels are regular; fæces are coloured. Inspection of the abdomen shows that it is enlarged. The branches of the internal mammary vein in the epigastric region are specially distended, but there are enlarged veins also in the hypochondriac, lumbar, and hypogastric regions. There is no evidence of ascites. The liver is distinctly enlarged. In the nipple line, deep dulness begins above at the fourth rib, and extends downwards $2\frac{1}{2}$ inches below the costal margin. In the middle line, the liver dulness extends $2\frac{1}{2}$ inches below the xiphi-sternum. The liver can be palpated easily; it is very firm in consistence, and over its surface, and at places on its otherwise sharp edge, little nodulations can be felt. The spleen is

much enlarged; it can be felt in the left hypochondriac region, and its anterior border can be traced to within an inch of the umbilicus.

Circulatory System is normal, with the exception of a soft blowing murmur, loudest at the mitral area (hæmic).

Respiratory System.—She has had a cough, spit, and occasional hæmoptysis.

Over the right apex, especially posteriorly, there is distinct percussion impairment, a somewhat bronchial character of breathing, and increase in the vocal resonance.

Since she came into the hospital she has had very little cough and no spit, so that the examination of the latter for the tubercle bacillus could not be carried out.

Urinary System.—No subjective phenomena. Urine amber in colour, acid, sp. gr. 1023, shows a slight trace of bile.

In these two patients we believe that we have examples of cirrhosis of the liver. Now, one of the first considerations which will strike you is that in both of them there is not only no diminution of the liver, but a distinct increase in its size. In this connexion it is to be remembered that this is the rule in liver cirrhosis of children. The explanation of this we shall defer until we are considering the course and prognosis of the disease. But although this sign is absent, we have, in the indications of portal obstruction which our patients present, distinct grounds for our diagnosis.

Let us now consider these signs of portal obstruction. These are, first of all, the enlargement of the spleen, explained by the great increase of pressure in the portal system. In both of our patients the splenic enlargement is marked. The notches or nicks which one looks for on the anterior border are, however, not distinctly felt. Next we have to notice the enlargement of the veins on the abdominal wall. This is due to the portal blood finding an obstacle to its passage through the liver, and so forming an anastomosing circulation

through the systemic veins. This anastomosis is mainly—in the stomach, between portal branches and œsophageal veins; in the liver, through the falciform ligament and perihepatitic adhesions between portal branches and diaphragmatic, internal mammary, and epigastric veins; in the mesentery, between portal branches and lumbar veins, and at the lower end of the intestinal tube, between the hæmorrhoidal veins and branches of the iliac and hypogastric veins.

In this way, in a well-marked case of cirrhosis of the liver we can see enlarged and often tortuous veins all over the abdominal wall. When these, as is sometimes seen, form a distinct ring round the umbilicus, the term "caput medusæ" is applied to the condition.

The importance of this anastomosis is very great, for it permits of the return of portal blood to the heart when through increasing obstruction in the liver the circulation through that organ is becoming interrupted. In this way we can explain the very striking improvement which we sometimes meet with in such cases. Thus some of you may remember the man L., who occasionally comes up to our ward. Over twenty years ago this man was in the Infirmary with ascites, and other symptoms of cirrhosis of the liver. He was then tapped nineteen times, some 400 ozs. of fluid being removed on each occasion. He then began to improve, and since then, although his liver is smaller than it was, and though he is thin and feeble, he has had alsolutely no fluid in his abdominal cavity, and has been able to move about in a quiet way.

Ascites is, as you can understand from what we have just been saying, a very common symptom of cirrhosis of the liver. In neither of our two patients has it been present to any appreciable extent whilst they were in the hospital.

In the little boy, however, as we are told by the doctor who attended him, it was present towards the end, requiring tapping.

Other symptoms of portal obstruction are gastro-intestinal catarrh, due to congestion of the mucous membrane of the alimentary tract, and as the result of this congestion hæmatemesis and melæna may occur. In neither of our cases is there any history of hæmatemesis or melæna, but in both there have been indications from time to time of gastro-intestinal catarrh.

Jaundice, which is not a common symptom of cirrhosis in the adult, is very common in the cirrhosis of children, and is probably due to a spread of the duodenal catarrh into the bile ducts. It is not usually severe; in our two cases there is only a slight coloration of the conjunctiva, and a slight trace of bile in the urine. The skin is hardly tinted, and the fæces are fairly normal as regards colour.

Pain in cirrhosis is not often very acute. The slight pains felt over the liver are probably due to perihepatitis. In the little boy patient the severe pains felt over the abdominal wall were, I believe, due to colic, the result of some tubercular enteritis. In the girl, the occasional attack of severe pain in the region of the liver, followed by slight jaundice, seemed to indicate some obstruction of the ducts, probably from inspissated bile. The emaciation which our patients show can easily be explained. We can readily understand that with portal obstruction the elaborating effects of the liver on nutritive material derived from the food will be interfered with.

Etiology.—The main cause of cirrhosis is some irritant, carried to the liver capillaries by the blood, and causing there an increased growth of the less specialized fibrous tissue, and corresponding disappearance of the more specialized liver parenchyma. Now, the blood going to the liver is of two kinds—portal blood and hepatic artery blood—and so the irritant may be carried by either of those channels. But it is evident that portal blood and hepatic artery blood are constantly intermingling, and so, although

we may be permitted to say that, for example, in alcoholic cirrhosis the irritant is carried mainly by the portal blood, and in syphilitic cirrhosis mainly by the hepatic artery blood, we yet must recognise this intermingling, and remember that in a large number of cases we cannot say definitely through what channel the irritant is carried. Alcohol, which is probably the most common cause of the disease in the adult, is also unfortunately a not uncommon cause of it in children. We are sure, however, that in neither of our cases has it been the factor, although, curiously, I find that in a case of cirrhosis in a girl lately in Dr Affleck's ward it has been undoubtedly the cause. In our cases also we have excluded syphilis, and in neither have we any ground to ascribe it to malaria.

But in dyspepsia of all kinds, and from any cause, we can readily understand that an important factor may be recognised. As the result of it, some abnormal and consequently irritating products will be carried by the portal system to the liver, and these will in time induce the disease. In the case of the boy there was, I believe, tubercular enteritis. In the case of the girl there was a history of long-standing dyspepsia. But, further, in the girl's case also there was distinct suspicion of tubercular lung mischief; and it is interesting to remember that the association in children of liver cirrhosis and tubercular disease has been again and again remarked.

To fevers the onset of liver cirrhosis in children has also been ascribed, the pathological explanation of this being the cell infiltration which takes place in kidneys, liver, and other organs during fever. In this connexion it is important to remember that in the boy patient the illness seemed to date from an attack of measles.

Taking all these etiological elements into consideration, I am inclined to think that in both of our cases the tubercular factor has been the most important. Whether, however, it

has acted indirectly through dyspepsia, or more directly through changes induced in the liver substance (tuberculous cirrhosis), I cannot say.

The course of liver cirrhosis in children is eminently unsatisfactory. There is little doubt that in cases which are slightly advanced recovery may readily occur if the cause can be removed. But when the portal obstruction has become so marked as to cause great enlargement of the spleen, ascites, or the establishment of a well-marked anastomotic venous circulation, the prognosis is very unfavourable. In time the patient gets more and more emaciated and feeble, and ascites, requiring repeated tapping, establishes itself. The direct cause of death may be some complication like peritonitis or pneumonia, or coma due to the presence of some toxin in the blood. A most noteworthy point is that in children the diminution in the size of the liver, so marked in adults, has very seldom been observed. This is explained on the ground that the course being more rapid in children, the organ has not in them sufficient time to contract.

As regards treatment, this is mainly dietetic and hygienic. Our patients were dieted on milk and starchy foods, with a little fish or chicken. When necessary, laxatives are indicated to relieve portal congestion. Both of our cases seemed to be improved by small doses of mercury; and in the boy's case, as already stated, we made use also of mercurial inunctions.

A CASE OF DYSENTERY TREATED BY DEËMETINISED IPECACUANHA.

GENTLEMEN,—John M., aged 29, a labourer, formerly a soldier, born and residing in Edinburgh, was admitted to Ward 30, on October 19, 1895, complaining of dysentery, and stating that he had been ill for six weeks. His family history is very good, and his home is comfortable, but as a labourer he is exposed to all sorts of weather. As regards previous illnesses, he had had scarlet fever as a child, and an attack of acute Bright's disease at the age of 20. He recovered from this completely, and then enlisted. As a soldier he went to India, and at Umballa he had an attack of dysentery which lasted for one month. After this he had ague several times, but his general health seems to have kept good, and he left the army about a year ago and took to work as a labourer.

Present Illness.—This began six weeks ago with diarrhœa, pain in the bowels, loss of appetite, and vomiting. The bowel evacuations were very frequent, eight or nine evacuations during the day, and often more at night. There was great straining, and the motions show mainly mucus and blood and a very little fæcal matter. Finding himself getting worse, he came to the Infirmary.

State on Admission.—Height, 5 ft. 6½ in.; weight, 8 st. 5 lbs.; used to weigh about 10 st. He looks thin and

anxious, and is very anæmic. He has little or no appetite; and after eating or drinking, vomiting usually comes on. He complains much of abdominal pain, and there is great tenderness on pressure over the descending colon and sigmoid flexure. With the diarrhœa there is great tenesmus, and a burning pain in the rectum. The dejections consist mainly of small quantities of muco-pus and blood, with little or no fæcal matter. We have repeatedly examined them for the "amœba dysenteriæ," but we have not succeeded in seeing it. The liver and spleen cannot well be percussed out, but we are of opinion that they are both slightly enlarged.

His circulatory, respiratory, urinary, and other systems are normal; his pulse is usually about 70 per minute, and his temperature about 99°. Here, then, is a case of dysentery, and we recognise what we often find in dysentery and in ague,—the tendency to recurrence as the result of some depressing cause. When we questioned our patient closely, he told us that although his health up to the commencement of his present illness had been very good, yet that when exposed more than usual he would have slight ague-like attacks, especially during the night, and also that his present dysenteric attack came on after a specially severe exposure to cold.

His progress and the modes of treatment employed can best be related by the following excerpts from the case-book:—

"On admission patient was put on milk and a little farinaceous food, and tinct. coto., ℥10, every four hours, was prescribed. As this in three days had not in any way modified the diarrhœa, bismuth subnit., 30 grs., thrice daily, was substituted; and large injections (3 or 4 pints) of warmed weak boracic solution were ordered to be given twice daily. The injections occasioned such severe pain that their administration had to be preceded by the introduction of ¼ gr. morphia suppository.

"*November* 8.—The treatment had not in any way affected either the character or frequency of the stools, the patient being called eight or nine times daily to pass, after much painful straining, a small quantity of bloody muco-pus. The bismuth was stopped, and pulv. ipecac., 5 grs., thrice daily, was prescribed. A douche of nitrate of silver (½ gr. to the oz.) was ordered to replace the injections of boracic solution, each douche to be preceded by the introduction of ½ gr. cocaine suppository. Two or three pints of this solution were allowed to pass into the rectum, the patient's pelvis being raised by a pillow, and care being taken to allow the fluid to pass in very slowly.

"*November* 13.—Very slight improvement in the diarrhœa. The patient still complains of pain in the abdomen, and great tenderness on pressure, especially over the sigmoid flexure and descending colon. The dose of ipecac. was increased to 10 grs. thrice daily.

"*November* 16.—As the administration of the ipecac. caused severe nausea and vomiting, which was not modified by giving previously ℥ 20 of liq. morph., it was stopped, and ℥ 40 of tinct. koromiko were given three times a day.

"*December* 2.—Since the ipecac. was stopped there has been a slight increase in the diarrhœa. The nitrate of silver douche and the tinct. koromiko, which had been increased to ʒj. thrice daily, had little good effect. There has been no change in the character of the stools, and the abdominal pain and tenderness is much the same as before. Pulv. ipecac. sine emetina, 10 grs., thrice daily, was ordered.

"*December* 6.—Little change as regards the diarrhœa, but the abdominal pain is not so severe, and tenderness on pressure is less marked. Dose of pulv. ipecac. sine emetina increased to 15 grs. thrice daily.

"*December* 9.—Patient feeling much better and anxious to

get up. The diarrhœa is not so severe, four or five stools daily. Abdominal tenderness hardly marked at all.

"*December* 11.—Patient was allowed to sit up, but felt very weak. Dose of pulv. ipecac. sine emetina increased to 20 grs. thrice daily.

"*December* 18.—Patient feeling much better. Diarrhœa much improved, two or three stools a day, and showing a little fæcal matter. No tenderness on the abdomen. Patient to get a little beef-tea and fish.

"*December* 26.—Improvement still maintained. Stools two or three daily, distinctly fæcal in character. Little or no blood.

"*December* 30.—Patient has only one motion in the twenty-four hours, the stool being well formed. He feels much better and is gaining in weight (8 st. 8 lbs.). The dose of ipecac. decreased to 15 grs. thrice daily.

"*January* 9.—On January 3 the nitrate of silver douche was stopped. Next day patient complained much of pain in the abdomen, and had four motions, the stools being watery and blood-stained. A return was at once made to the nitrate of silver douche, the patient was ordered to keep his bed, and the dose of ipecac. sine emetina was increased to 20 grs. thrice daily.

"*January* 19.—Patient is very much better. The bowels are moved once or twice daily, the stools being of normal colour and consistence. He is getting ordinary diet and is going about in the ward. With the exception of a slight remission on January 23, probably the result of exposure, patient's progress towards convalescence was very satisfactory. He was discharged on February 4, his weight being 8 st. 12 lbs. He was recommended to take a holiday in the country, to continue taking the ipecac. powders in 10-grain doses twice daily along with a tonic, and to report himself in two or three weeks."

There has been some difference of opinion as regards the

value of deëmetinised ipecacuanha. By some it has been found of such slight efficacy that it has been asserted that the antidysenteric effects of ipecacuanha are mainly due to the emetina. In the hands of others, however, it has been found of very great value (Kanthack and Caddy, *Practitioner*, June 1893). In our patient there is no doubt as to its having been of the very greatest use.

AORTIC ANEURISM;

WITH SPECIAL REFERENCE TO SENSITIVE AREAS ON THE SKIN.

History and Description of a Case.

Gentlemen,—D. R., aged 64, a joiner, formerly a soldier, was admitted to Ward 30 in the Royal Infirmary on March 9, 1895, complaining of pains in the chest and back.

His family history was fairly good, and indicated no tendency to any particular disease. In early life he had been a soldier, and he had gone through all the hardships of the Crimean War; in later life he had been a joiner, and had had occasional heavy lifts. With the exception of specific disease in his soldier days, he had had no previous illness. He had always been very temperate, well fed, and well housed. His present illness began about eighteen months ago, when he noticed that he was subject to a dull aching pain in the back of his neck. About six months ago he noticed also a pain in his chest. Those pains were of a dull boring character, and were always brought on by exertion. Recently he had also noticed some loss of power in the right arm. In spite of these pains the patient had been able to continue his work till a week ago, when they became very severe. Applying for relief to a medical man, aortic aneurism was diagnosed, and he was recommended for admission to the Royal Infirmary.

His height is 5 feet 6 inches, his weight 10 st. 7 lbs., but

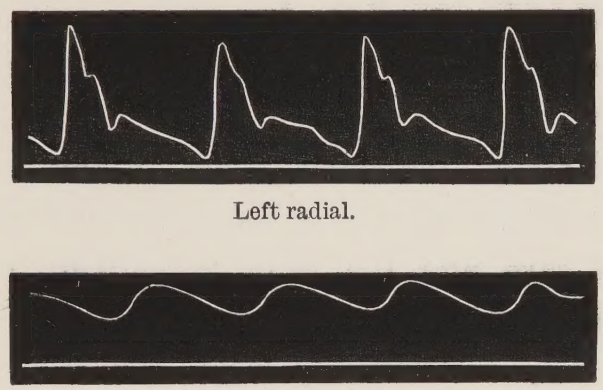
he states that he used to weigh over 11 stones. His development and muscularity are fairly good for his age, and, with the exception of slight œdema of both ankles and of the right wrist, there are no very obvious morbid appearances. His appetite is good, and he states that he has no difficulty in swallowing. His digestion is also good, but when the chest pain is severe he states that he is troubled with flatulence. On physical examination his digestive organs appear normal, except that the liver is slightly enlarged.

He makes no complaint of dyspnœa, but states that he has slight palpitation now and again. He has pain over the precordia and in the upper part of the chest generally, specially on the right side. He has also some pain in the back. As already stated, these pains are all apt to be brought on by exertion. On physical examination the heart is found to be enlarged, its impulse beat being in the sixth interspace, three-quarters of an inch external to the nipple line. Over the manubrium sterni and over the first interspace on either side of it, but more especially on the right side, pulsation can be made out. On palpation over this area the pulsation is recognised as expansile, and very heaving and powerful. Each beat directly follows that of the heart, and a distinct diastolic impulse can be made out. Percussion reveals great enlargement of the heart and marked dulness over the manubrium and parts adjoining, where, as already stated, pulsation can be seen. On auscultation over the base of the heart and over the pulsating area a slight systolic murmur is heard, and the second sound is pump-valve in character. The pulse, about 70 a minute, varies in character in the two radials, as seen in the tracings. It is noted that the right carotid is also weaker than the left.

The breathing, 22 a minute, is costo-abdominal in type. There is no cough, no hoarseness of voice, no stridor. Physical examination of the lungs reveals nothing abnormal,

but "tracheal tugging" is very well marked, and careful examination shows slight displacement of the trachea towards the right side.

FIG. 1.



Left radial.

Right radial.

Sphygmographic tracing from case of D. R.

The integumentary and urinary systems are normal. As regards the nervous system and special senses, all that need be said here is that the right pupil is much larger than the left. Both pupils, however, react readily to light and accommodation.

In this case we had no difficulty in diagnosing aneurism of the aorta, with enlargement of the heart, especially of the left ventricle. Any valvular disease we believed, if present, to be very slight. The aneurism seemed to involve mainly the ascending and transverse parts of the arch. Although pulsation and dulness were present to the left of the sternum, we did not believe that the descending part of the arch was involved to any extent, because there was no dulness in the left interscapular region. The enlargement of the heart we associated with the aneurism. We could not ascribe it to any aortic valvular disease, as the pump-valve second sound negatived regurgitation, and the absence of any marked systolic murmur, and the well-filled arteries of the left side, negatived obstruction. Some authorities hold that aneurism *per se* entails extra work upon the heart, and so causes hypertrophy; others deny this; but whether or not, we may be

sure that the conditions which lead to aneurism, namely, atheroma, strain, etc., lead also to cardiac enlargement. You all know that in a case of difficulty of diagnosis between aneurism and mediastinal tumour, an enlarged heart is a point strongly in favour of the former.

As to pressure symptoms, we believed, in the first place, that there was no pressure upon the gullet. The slight œdema of the legs, the enlargement of the liver, and the slight œdema of the right arm, might be ascribed to pressure on the vena cava and the right innominate vein, but the absence of distinct venous dilatation anywhere precluded the idea of the existence of any marked pressure here. As regards the trachea, we believed that the fact that it was slightly pushed towards the right, and the marked "tracheal tugging," indicated that it was involved, but in the absence of stridor, etc., we thought that it itself was not to any extent compressed. There were, however, in the smallness of the right radial, brachial, and carotid pulses distinct indications of involvement, and in connexion also with the nervous system there was distinct evidence of pressure. To these I wish now to refer in some detail.

The Difference in the Pulses.

The small pulses of the right side might be due to the innominate artery either being pressed upon by the aneurism, or to its being partially closed by a clot at its origin from the aorta. The more sloping upstroke, curved summit, and gradual downstroke, shown by the sphygmographic tracing, with the almost complete disappearance of the predicrotic and dicrotic notches, could be explained on the theory that the dilated and expansile aneurismal sac acted like the elastic indiarubber bag of a spray producer.

But, you will ask, if the ascending and transverse portions of the arch in this patient were dilated and formed a large elastic sac like the bag of a spray producer, why did not the

tracing from the left radial show similar appearances? At the time I thought that this might be explained by supposing that the aneurism sprang from the arch at a part close to the origin of the innominate artery, and that the greater part of the transverse portion of the arch was not dilated. But a post-mortem examination (the man died from slow rupture into the pericardial sac three days after his admission) showed that the whole of the ascending part of the arch outside the pericardium and the whole of the transverse portion were involved, and that the left subclavian arose from a part as dilated as was that from which the innominate arose. Hence the marked difference in the character of the tracings from the right and left radials is to be ascribed to the opening of the innominate being narrowed by a clot, and that of the left subclavian being perfectly free.

The Nervous Symptoms.

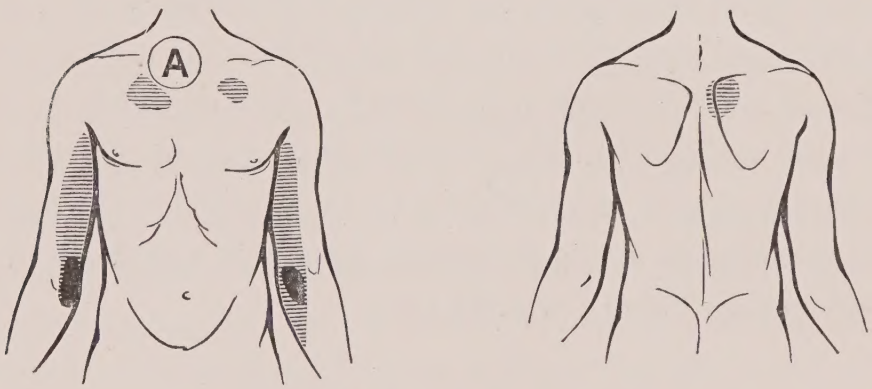
As regards nerve structures involved, we have seen that in this patient the right pupil was dilated, indicating pressure upon the sympathetic on the right side, and irritation of the fibres which pass up to the pupil from the cilio-spinal region. There were no indications of involvement of either of the recurrent laryngeals, there was no hoarseness, and the vocal cords moved freely. But I wish specially to refer to the parts where pain, numbness, or other disturbances of nerve function were felt.

As you are aware, the pains about the chest, down the arms, etc., which are so often complained of in aneurism, are usually ascribed to pressure of the aneurism upon branches of the brachial plexus and intercostal nerves, but they are much more appropriately to be referred, as Mackenzie,[1] and notably Head,[2] have shown, to peripheral irritation of the various segments of the spinal cord.

[1] *Medical Chronicle*, August 1892.
[2] *Brain*, vol. xvi., 1893.

In this patient we had hardly time, owing to his rather sudden demise, to mark out the painful and sensitive areas sufficiently carefully, but we have two other patients with aortic aneurism in the ward, and in them we have done so. I shall first, therefore, refer to them, and postpone alluding to this case till afterwards.

Fig. 2.

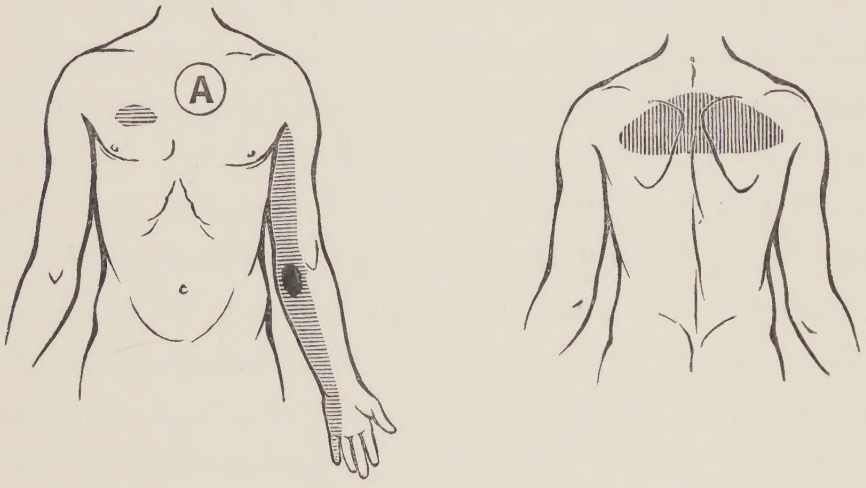


Thus Fig. 2 represents a patient D., at present in the ward. The area (A) represents the position of his aneurism as demonstrated by percussion. The shaded areas represent the parts where pain is specially felt, and where the skin can be shown to be specially sensitive to the prick of a needle. In this case we believe that the painful areas in the front of the chest, and the specially sensitive spots at the elbows, correspond to irritation of the first dorsal segment; that the painful areas along the inner aspects of the upper arms correspond to irritation of the second dorsal segment, whilst that the painful spot in the right interscapular region posteriorly corresponds to irritation of the third dorsal segment. In this case the aneurism mainly involves the ascending arch and innominate, and, as seen, the tender areas are on both sides anteriorly.

In the second case, R., Fig. 3, in whom the aneurism (A) seems to involve mainly the transverse part of the arch, the

painful chest area is anteriorly on the right side alone, and seems to correspond with the third dorsal segment. But the left arm shows, in addition to the specially sensitive spot at the bend of the elbow, an area of tenderness along its entire inner aspect. This extends right down to the fingers,

FIG. 3.



where it corresponds to the distribution of the ulnar nerve, and indicates irritation of both first and second dorsal segments. Posteriorly there is a large area, symmetrical on either side. It is specially painful and sensitive, and seems to correspond with the second, third, and fourth dorsal segments.

In the patient D. R., the subject of this lecture, we had not, as already stated, time to mark out those areas carefully. It was noted, however, that in addition to painful areas on the anterior and posterior aspects of the thorax, in addition to complaints of pain down his arms, and of occasional loss of power in his right arm, he complained of a painful spot at the back of his neck, about the vertebra prominens. This seemed to correspond with the posterior distribution of nerves from the first dorsal segment. In studying those areas in all those cases we endeavoured to discover if any

precise connexion existed between the locality of the aneurism on the arch and the painful or sensitive areas. We could not, however, come to any more definite conclusion on this matter.

Treatment.

Our cases of aneurism are being treated by absolute rest, low diet, and large doses of iodide of potassium, with the addition of a little chloral if iodism tends to show itself. As regards the mode of action of the iodide of potassium in such cases, there is great difference of opinion. According to some, it acts by lowering the blood-pressure; according to others, by increasing the secretions, inspissating the blood, and so promoting clot formation; whilst a third view is that it acts by causing thickening and contraction of the sac. As to its power of lowering the blood-pressure, all our observations negative this. With the sphygmomanometer we have found that no marked lowering of the pressure occurs as the result of the use of large doses of iodide of potassium, and you will remember the case of the man X. (specific disease), in whom we gradually increased the dose of the iodide till he was taking between two and three drachms a day, and in whom careful sphygmomanometric examination showed no lowering of the blood-pressure.

The view that iodide of potassium causes inspissation of the blood we also do not feel inclined to accept, for in several of the patients in our ward who are taking iodide of potassium we have tested this, and we found that the specific gravity of the blood was not increased. Whether or not, however, the iodide has a special effect in causing the blood to clot more rapidly we are unable to say.

Lastly, as regards the view that the benefit of the iodide is to be ascribed to its affecting the wall of the aneurismal sac. It is evident that as the main effect of the drug is supposed

to be to promote absorption, any beneficial effect which it exercises in aneurism must be the result of its tending to remove whatever causes the sac wall to yield, and not of its tending to increase what might cause the sac wall to contract. Hence some hold that the iodide is useful only in specific aneurisms, but on this point we cannot speak with any certainty.

A CASE OF EPILEPSY PRESENTING SOME PECULIAR FEATURES.

GENTLEMEN,—R. J., aged 13, a schoolboy, residing in Edinburgh, was admitted to Ward 30 on January 17, 1893, complaining of fits, from which he has suffered for the last eighteen months.

History: Family.—He is one of a family of five. His parents are both alive and well, and the only points which seem noteworthy are that his father seems to be a rather nervous man slightly addicted to alcohol, and that there is a phthisical tendency in the father's family.

History: Personal.—His mother states that he has had no illnesses previous to his present one. We asked if he had had any convulsive attacks in infancy during dentition, but found he had had none.

Present Illness.—Eighteen months ago, when playing in Leith Street, he was knocked down by a passing cab, the shaft striking him on the forehead above the left eye. He states that he was stunned, and that when he came to his senses he found himself in a chemist's shop. He then walked home. This took place on a Thursday. On the morning of the following Saturday he took his first fit, and felt very ill all that day. About a fortnight afterwards he had another fit, and he states that they were occurring at similar intervals till three weeks ago. Since then he has been having a fit every night about ten o'clock, just as he is dropping off

to sleep. Last Friday night (January 13) he had five fits, but he was able to stop three of them by grasping his leg in the manner described below. He describes the fits as follows:—He is made aware that one is coming on by a tickling feeling in the ball of the great toe of the left foot. He cries out directly the tickling comes on, and says he cannot help crying out. He grasps the leg above the knee with both hands as tightly as he can, and often he has got his brother, with whom he sleeps at night, to grasp it too. At times he is able in this way to prevent the tickling passing up the leg, and so to prevent the fit coming on. At other times he is unable to do this; the tickling extends up to his head, and he becomes unconscious, and remembers nothing more till he awakes from sleep some hours afterwards. He has never bitten his tongue, or at least it bears no marks of having been bitten; and he has never observed blood about his mouth or on his pillow in the mornings. From his mother's description, however, we had no doubt from the first that the fits were distinctly epileptic, and since his admission we have had the opportunity of seeing him in several paroxysms, so that we have no doubt upon this point. Let us, however, in the meantime confine ourselves to the phenomena which we met with in his case on his admission.

State on Admission.—Height, 4 ft. 10 in.; weight, 6 st. 9 lbs. He is a well nourished and developed boy, of good muscularity. A small abrasion can be seen about two inches above the left eye, which he says has been there since his accident. His temperature and pulse-rate are normal. His expression is happy and contented, and he has a stammer in his speech, which has, he says, always affected him.

Nervous System.—Voluntary motor functions normal.

Skin sensibility to touch, temperature, and the faradic current is normal all over. On the left leg or foot it shows little, if any, difference from the right, the only point worthy

of note being that, when testing the sensibility of the sole of the foot by tickling that part, the patient cried out on one or two occasions, and grasped his leg, feeling, as he expressed it, the twitching which is apt to pass into a fit.

Skin Reflexes.—On admission, the plantar, cremasteric, epigastric, abdominal, and scapular reflexes were all found to be well marked,—it was thought, on the whole, rather more on the left side.

Deep Reflexes.—The knee-jerk was found distinctly more marked than usual in both legs; and in both ankles, on tapping the tendo Achillis, distinct reflexes could be elicited, and a slight tendency to ankle clonus could be made out. With the deep reflexes, as with the superficial, we were careful to test if any relative increase could be made out on the left side, but we could not altogether satisfy ourselves that it was so. More, however, will be said about this by-and-by.

Another reflex which we examined on his admission was the faucial reflex. As you all know, on tickling the fauces a reflex contraction of the parts results, more or less marked in different individuals. Now, it has been said that this reflex is well marked in epileptics, corresponding to the increased irritability of the nerve centres existing, or supposed to exist, in that disease, and that the reflex irritability of the parts there may be useful as a guide to the efficiency of the bromide of potassium.[1] That is to say, that with the administration of the bromide the number of the fits and the reflex irritability of the fauces will be found to diminish together. Here, then, was a case in which a knowledge of the reflex irritability of the fauces would be specially interesting, for the fits had been becoming more and more frequent, and the patient had not been taking any bromide. On examining into it, however, we found that it was conspicuously absent; as one of our students

[1] Compare Osler, *Principles and Practice of Medicine*, p. 956.

remarked when this was demonstrated, our patient would make a splendid subject for laryngoscopic examination. This absence of the faucial reflex was, however, quite what I expected. In most cases of epilepsy which I have examined I have found it so, whether the patients have been taking the bromide or not.

Special Senses.—These are all normal. With the ophthalmoscope no abnormality can be discovered in the fundus.

Mental Functions.—These also are normal. His mother states that he is an average boy as regards his school lessons, and that she does not think that he has suffered there from overpressure. He has a stammer in his speech, but this has been present all his life.

Urinary System.—About 50 ozs. daily, acid; sp. gr. 1020; no abnormal constituents.

Such, then, is our case. You will, I think, agree with me in considering that it presents many interesting features. These I now propose to consider in detail, and I shall begin by discussing with you the "aura" of which this case presents so distinct an example. Epileptic auræ may be sensory, motor, special sense, psychical, or visceral, but in this case our only difficulty seems to be to decide whether the aura is sensory or motor. He tells us, as you will remember, that it begins by a tickling sensation in the ball of the left great toe, which spreads up the leg. In this way it might be regarded as a sensory aura. But he tells us—what, indeed, we have had the opportunity of observing since his admission—that it is associated with a twitching and contraction of the muscles of the foot and leg, and so we might believe that it was a motor aura, and that the tickling sensation may be due to muscle rather than cutaneous sensibility. Which of the two it is we cannot well say, but there is no doubt that sensory impressions tend to excite it. It will be remembered that tickling the soles of his feet, so as to test the sensibility or excite the plantar reflex, was found by the clerk who took

the case to arouse the aura. Further, although now in our visits to his bedside in the ward we find that acting in this way does not so readily induce the aura sensation, Dr Baines informs me that if at night he awakes our patient and tickles the sole of the foot, especially about the ball of the great toe, the aura sensation is at once induced so strongly that the patient feels that it is only by a timely and firm grasp of his leg that a true paroxysm can be prevented from coming on. It is interesting to remember that many cases presenting similar peculiarities as regards the nature of the aura and the effect of skin irritation in provoking an epileptic paroxysm have been reported. As examples of these I quote the following from a paper by Dr Ogle[1]:—

A man, aged 21, who had had syphilis, but who had had epilepsy before the syphilis, was having several attacks in the day, and noticed that each attack was preceded by an aura, a sensation as of blood rushing up from the left side of the chest and left arm to the head. Dr Ogle found that suddenly jerking the left arm, or roughly handling the left shoulder, or pinching the skin of these parts, could at once induce an attack. He also observed that to induce an attack a second time, it was necessary that an hour or two should have elapsed after the first one, as if to permit an accumulation of force.

Another case, also reported by Dr Ogle, was that of a boy who had a fatty tumour over the upper part of the right scapula. Whenever the tumour was handled, however lightly, the boy became unconscious, and his body assumed the position of intense episthotonus, this position being maintained for half a minute, or longer if pressure were continued.

These observations remind us at once of the "epileptogenous" areas first discovered and described by Brown-Séquard. As you know, in the case of the guinea pig rendered epileptic artificially—*i.e.*, by section of portions

[1] *Lancet*, 1874, vol. i., pp. 615 and 651.

of the spinal cord, etc.,—it has been shown that the epileptogenous area is for the most part the skin about the angle of the jaw. What has caused the "epileptogenous area" in our patient to be the skin over the ball of the left great toe we cannot well say. We have tested the parts carefully for any indications of altered sensibility as regards touch, temperature, and the faradic current, also for any indications of altered trophic function, but beyond what has been already described we have discovered nothing.

Next, I wish to say a little as regards the spread of the aura. As already stated, the boy tells us that if he is unable to stop the tickling sensation by firmly grasping the leg, it (the sensation) rapidly passes up to his head, and then he loses consciousness. On questioning, however, more closely, he tells us that after he feels it in his head it seems to pass to his left arm and then to his right arm, after which he knows nothing; and he does not know when his right leg becomes affected.

Remembering, however, that the aura is simply a part of the epileptic paroxysm, we have obtained further information on this point by observing the sequence of events during the onset and course of a paroxysm. When, for example, he has found that grasping his leg has been of no avail, he lets go his leg with a sort of cry, and we notice that his muscles are all contracted in the so-called tonic stage. We do not get much information from this; but if we watch for the onset of the clonic stage we can discover that it begins first in the legs, then spreads to the arms, and, lastly, to the face. I am therefore disposed to believe that the spread of the epileptic nerve disturbance in this case is the usual one, viz., from the leg to the thigh, then to the opposite limb, then to the arm of the same (left) side, then to that of the opposite (right) side, and, lastly, to the face and head.

As regards the meaning of the cry, so often present in epilepsy, there is a difference of opinion among physicians.

One theory is that it is in a way voluntary, indicating fear or surprise; while another is that it is, like the aura, a part of the fit, and caused by a convulsive contraction of the thorax and narrowing of the glottis. It has been argued against the first theory that patients have no recollection afterwards of having cried out, or experienced fear or surprise; but the upholders of this theory point out that after a paroxysm the patient's memory of its immediate antecedents is apt to be obscured. Probably, however, the other theory is the better,—the associated contraction of the thorax and narrowing of the glottis being the result of the epileptic nerve disturbance involving the phonation centre in the cerebral cortex. In our patient the fits have, since his admission, been usually accompanied by a cry, but he does not seem to remember about this afterwards.

The attacks themselves in this case present no very unusual features. We notice during the tonic stage the powerful, and, as it were, steady contraction of the muscles, the initial pallor of the face, the converging of the eyes, the dilating of the pupils. Then follows the dusky suffusion of the face by the respiratory stoppage, and the clonic stage manifesting itself by the jerking of the limbs, the convulsive twitchings of the muscles of the face, the frothing at the mouth, etc. As compared with average epileptic paroxysms the attacks are not severe ones; he does not bite his tongue, his recovery is rapid, and the flea-bite-like petechial hæmorrhages,—the indications of intense venous congestion,—which one so often finds on the forehead after a paroxysm, have never been seen. Shortly after the fit he passes, however, a large quantity of rather high-coloured urine. On several occasions we have examined this; we found it acid, with a specific gravity of 1022, and clear.

Remembering that temporary albuminuria or glycosuria may follow an epileptic paroxysm, we have tested it for albumen and sugar at those times, but have found none. On

adding to it a little caustic potash, however, we have observed that this post-paroxysmal urine shows a distinctly greater cloudiness (phosphates) than the urine passed at other times.

Let me next draw your attention to the fact that in our patient the paroxysms are mainly nocturnal. As you are aware, this is the case with many epileptics; and, as Dr Féré states,[1] this fact has served the upholders of the theory that epilepsy is associated with congestion of the nerve centres, as well as those who believe that it is associated with anæmia, with what they have regarded as evidence,—the partisans of the congestion theory pointing to the prone position, the partisans of the anæmia pointing to the physiological condition of the nerve centres during sleep.

In the case of our patient the attacks, as just stated, came on for the most part during sleep at night, but it is interesting to remember that on one occasion (23rd January), when the nurse, finding him even more mischievous than usual, had sent him to bed, he had between the hours of 4 and 10 P.M. eight attacks of the aura. None of them, however, passed into a regular paroxysm, and, of course, in such circumstances it is impossible to decide how much what is understood as "free will" had to do with their production. It is noteworthy, however, that he has never had a paroxysm when up and going about.

But what is no less interesting is the time of the night at which paroxysms are most apt to occur. Dr Féré, from a great number of cases, has shown in a most interesting manner that their occurrence is most frequent about 9 o'clock at night, or 3, 4, or 5 o'clock in the morning,—that is to say, about the times which follow going to bed or precede getting up. As you will remember, these are precisely the times when sleep is least likely to be profound, and when dreams, the results of partial cerebral activity, are also most frequent. In our patient the fits came on before his admission, as he explained

[1] *Les Epilepsies et les Epileptiques*, Dr Ch. Féré, p. 312.

it, just as he was going off to sleep; since his admission they have occurred mostly about 2 or 3 A.M.

In our patient we have further endeavoured to obtain information as regards the state of his nervous system during the intervals, and we have carefully looked for any alterations in this at the period preceding and succeeding a paroxysm. As you can understand, information of this kind would be of great value in helping us to a knowledge of the pathology of epilepsy, and careful investigations of this kind would well repay the trouble. What we have been able to do at present has not been very much; but such as it is, let me now describe it to you.

At certain regular periods, viz., at 10 A.M., 2, 6, and 10 P.M., and at 2 and 6 A.M., and when opportunity offered during the pre- and post-paroxysmal states, we ascertained the pulse-rate, temperature, voluntary muscular power by the dynamometer, the state of the cutaneous and deep reflexes, and the state of the electro-sensibility and electro-contractility to the faradic current in the legs and feet.

We found the pulse-rate and temperature to show great regularity. No difference in them have been noticed in the pre-paroxysmal stage, but a rise has been noticed in the temperature immediately after the fits. Thus in one instance when the fit occurred at the usual time, 2 A.M., and when, as his chart shows, the temperature is about its lowest,[1] his temperature was found to be 99° F., pulse 64, and respirations 24; whilst an hour after, the temperature was 97°, pulse 88, and respirations 16.

As is known, the voluntary motor power, as ascertained by the dynamometer in the hands, is diminished in the post-paroxysmal stage. Our patient, whilst ordinarily registering 70 R. and 50 L., after the attack usually registers about 45 R. and 35 L.

[1] Compare Féré, *Les Epilepsies et les Epileptiques*, p. 313; also Bevan Lewis, *Med. Times and Gazette*, March 1876.

As regards reflexes,[1] our observations seem to indicate a slight increase in the pre-paroxysmal, and diminution in the post-paroxysmal stage, and as regards electro-sensibility and contractility a diminution in the post-paroxysmal stage.

Diagnosis.—On this subject, what we have now to do is to make up our minds whether we are dealing with a case of idiopathic epilepsy,—of an epilepsy in which, supposing we had at present an opportunity of examining the nerve centres, we should find practically nothing to account for it,—or a case in which, as one of the results of the injury, we should find some gross lesion. Remembering that our patient received a severe injury to his head some eighteen months ago, and that his mother dates his epilepsy from this time, and remembering also that each attack begins with a distinct aura in the left foot, we might not unreasonably suppose not only that there existed such a lesion, but that it existed at a certain part of the brain.

I have come to the conclusion that there is no gross lesion, however, for the following reasons:—No trace of injury nor tenderness can be detected on examining the head at any part; and although the aura always begins in the ball of the left foot, a careful examination of the limb shows no alterations—motor, sensory, reflex, or trophic—such as one might expect with a gross lesion. I have also known of other cases in which as distinct a peripheral aura was present as exists in this case, and no gross lesion of the brain was found. He has, further, no headaches nor vomiting, and the examination of his eyes by the ophthalmoscope reveals nothing abnormal. Lastly, epilepsy due to a localized lesion is more frequently a partial than a general one, as is the case with our patient.

[Concerning the value of these two last considerations, let me remind you, however, that we cannot speak very

[1] Compare Féré, *Les Epilepsies et les Epileptiques*, p. 157; also Beevor, *Brain*, April 1882.

confidently,—more especially since, as many of you will remember, we have had in Ward 30 two cases illustrative of the opposite of this. One was the case of a man, B., who had developed attacks of general epilepsy as the result of a rapidly-growing sarcoma of the left cerebral hemisphere, and in whom repeated ophthalmoscopic examination revealed no changes whatever in the retina. The other was the case of the man, S., admitted for hemiplegia of the right side—face, arm, and leg—and in whom we diagnosed a syphilitic thrombosis in the left internal capsule. This man, shortly after his admission, developed epilepsy, which we recognised as post-hemiplegic, and as being due to the irritation of the area of softening in the internal capsule. Although the lesion was a localized and not a very extensive one, the epilepsy was general. Many other examples could be given, but these are sufficient to demonstrate that the absence of optic nerve changes does not negative the presence of gross cerebral lesions, and that the fact that the epileptic paroxysms are general does not in any way negative the existence of a localized brain lesion.]

But now that we have concluded that there is no gross lesion in this case, what importance are we to assign to the injury in connexion with the causation of the disease? Recognising that an injury such as this boy received must certainly have had a prejudicial effect, I am yet inclined to think that the main factors in the production of his epilepsy have been constitutional ones. In his father's family there is a marked phthisical tendency, and his father seems to be a nervous and irritable man, inclined at times to alcoholism.

Further, the boy's mental organization is certainly not a very sound one. Nurse tells us that she has great difficulty in keeping him out of mischief in the ward, and his mother told me that he has all along at home been in the habit of receiving severe and frequent thrashings from his father. Whether, for such a state of affairs, the nervous organization

of the boy or of the father is the more to blame, we need not concern ourselves; we have ample grounds for concluding that our patient is to be regarded as neurotic. Again, we have to remember that the age at which the disease began is that of ordinary idiopathic epilepsy, and, lastly and most important, that there is a distinct history of an "aura" having been observed at a date antecedent to his accident.[1] I am therefore inclined to ascribe his illness mainly to constitutional causes; these in the absence of his injury would probably have produced the epilepsy, whilst the accident, without the constitutional predisposition, would probably have had no such effect.

Prognosis.—Although we may reasonably hope that our patient will live for years, and that periods of freedom from attacks will from time to time show themselves, we cannot but admit that as regards the disease itself we cannot speak very favourably. Had we been able to ascribe it more to injury and less to constitution, we could, of course, have hoped for more from time and care. As you know, frequency rather than severity of paroxysms gives the graver prognosis, and in our patient the attacks, up to the time of his admission, have been increasing in frequency. By many physicians the markedness of the aura is looked upon as favourable; and there is no doubt that when, as in our patient, its occurrence gives an opportunity for arresting the fit, it is a still more favourable sign. Nocturnal attacks are by some regarded as more favourable than diurnal, as being less likely in time to induce that impairment of the intelligence so common in epileptics.

Treatment.—This may be divided into the treatment—(*a*) of the paroxysm, and (*b*) of the interval.

(*a.*) For the paroxysm we provided our patient on his admission with a strap round his left leg above the knee,

[1] This information we obtained some days after his admission, as the result of careful inquiry.

which he could tighten when he felt the aura. He has discarded this, however, as he says that he has discovered that he can do better by grasping the leg tightly with his hands. If a scientific explanation were asked for this, it might be said that powerfully grasping his leg has a derivative effect on the nerve force which is being generated, and prevents an explosion of epilepsy; just as firmly grasping the arms of the dentist's chair has a derivative effect on the nerve force generated by a painful tooth-extraction, and prevents explosions of other kinds.

[Such modes of arresting a fit are not uncommon. Forced flexion, forced extension or twisting of a limb, pinching a finger or toe, or the skin of the ear, etc., have all been found in certain cases to act more or less efficiently.]

In such a case the nitrite of amyl inhalation might be tried when the aura is felt, but hitherto we have not used it.

The attack having recurred, we let him work through it. We do not require to put any protecting body between his jaws, as he does not bite his tongue, and we let him sleep after the fit, recognising that its occurrence means exhaustion of the nerve centres, and a need for rest.

(*b.*) In the intervals he has been taking the bromide of potassium in 20-gr. doses thrice daily, and the result is that the fits are much less frequent. We propose in the meantime to continue this.

A CASE OF SPINAL INJURY.

GENTLEMEN,—D. G., æt. 28, a miner, was admitted to Ward 30 on November 6, 1893, complaining of muscular weakness, and of a feeling of coldness and numbness in the legs, dating from a severe injury to his back sustained on December 6, 1892.

HISTORY: *Family.*—His father, aged 59, is a healthy man, but has suffered at times from dropsy. His mother died of cholera; and he has six brothers and one sister, all alive and well. He has always had plenty of good food, and has never drank so much as to prevent him going to his work. He used to smoke as much as 6 ozs. in the week, but for the last five years has not smoked more than 2 ozs. At home he is quite comfortable, but he is much exposed when at work, especially as latterly he has worked in a Welsh mine at a very great depth. He has always been strong and healthy, and had no accident till the one now to be referred to. This happened on December 6, 1892, when, just as he was beginning work, a large mass of coal fell on his back, and he remembers nothing till he recovered consciousness nine days afterwards. On the tenth day after (December 16) he was admitted into Llanelly Hospital, where he was told he had broken his back, and that three of his ribs had also been broken. At that time he states that he suffered intense pain over the small of his back, which shot down both his legs and up to his head, and also a severe headache, which lasted for months. He had also lost power completely in his legs, and he had no feeling in

them, nor, indeed, from his waist downwards. He states also that his arms were paralysed, and that his breathing was very laboured. He had no loss of feeling in his arms; he could eat his food fairly well, but he experienced a difficulty in speaking, and he noticed that his sight was not so good as it had been. He had complete loss of control over his bladder and rectum. He remained in hospital at Llanelly for three months, during which period he made some improvement. The power in his arms returned after about a month, and about this time also he recovered control over his bladder and rectum. He then returned home, and was treated with the faradic current, which he says relieved considerably the pain in his back, and seemed to restore the feeling in his legs to some extent. In May last he came back to Scotland, at which time his legs were still paralysed. On one day in August last he noticed that there was more feeling in his right leg, and he found that he could move his right thigh on his abdomen. At this time he was taking strychnia by advice of his doctor, and he seems on one occasion to have taken an overdose by some mistake of his own. He described to us how, immediately after he had swallowed this, he experienced the most terrible tremors through his body,—his jaws were locked, and he lost both vision and the power of speech, though his mind was quite clear. Emetics were administered, but did not make him vomit, and gradually his bad symptoms improved, but he says that ever since he has had tremors through his muscles from time to time. He, however, continued gradually to improve, and the power in the left leg returned sufficiently to enable him to walk with the aid of two sticks. He says, however, that trying to walk always causes pain over the lower part of his back and down his left side. About four months ago he had severe pain in his stomach, which caused him to vomit; he also had severe palpitation and giddiness after taking food. About this time, too, he noticed a feeling of formication on the front of

his right leg, and this has occurred several times since. He then sought admission to the Infirmary, and was taken into Ward 30.

State on Admission.—Height, 5 ft. 4 ins.; weight, 8 st. 11 lbs., used to be 10 st. 4 lbs. Development good, though muscles are soft and flabby. The muscles of the legs appear the most reduced. The spine of the first lumbar vertebra is prominent, and there is pain on pressure over it, and, indeed, over all the lower dorsal, lumbar, and sacral spines.

NERVOUS SYSTEM: *Sensory.*—He has an aching pain over the lower part of his spine and left side, and a feeling of coldness, specially over the sacrum. There is a feeling of numbness over the front of the left thigh, disappearing over the front of the knee, but becoming marked again over the front of the leg and the dorsum of the foot. It is also marked over the dorsum of the right foot and the front of the right leg. The sensibility to touch, pricking, and temperature is lost over these parts, and careful testing shows that over the lateral and posterior aspects of both legs these sensibilities are also impaired. But it is noteworthy that the sensibility in the soles of the feet is much better marked than in the legs.

Motor.—Voluntary motor power over the muscles of the thighs is fairly good; over the muscles of the legs, on the other hand, it is markedly lost. This can be seen very well when the patient sits on the edge of his bed with the legs allowed to hang down. It is then seen that, although the muscles of both limbs are much smaller than normal, the leg muscles are much more reduced than those of the thigh, and that when he is asked to move the foot on the leg at the ankle joint, little or no movement occurs, while when he is asked to move the leg on the thigh at the knee, the movements, both of flexion and extension, are briskly performed. In cases like this it looks almost as if these latter movements were more briskly performed than in a normal individual, as,

indeed, the thinned leg must be lighter to move, and so give specially easy work to the thigh muscles to perform. When we examine these weakened leg muscles more particularly we find that the tibialis anticus and peroneus longus and brevis are specially affected, in consequence of which he cannot dorsiflex, abduct and adduct his foot. But the muscles of the back of the leg are also affected, his power of pointing the foot being limited. These voluntary motor impairments are most marked on the left leg.

Reflexes: Superficial.—Plantar are present on both sides, specially on the right. Cremasteric are also present, and abdominal are well marked. *Deep.*—The ankle-jerks are well marked on both sides, and on both sides clonus can be elicited. The knee-jerks are markedly absent on both sides, and there is not the slightest trace of clonus, either on tapping the patellar tendon with the leg semiflexed, or on suddenly and forcibly drawing down the patella when the legs are straightened out.

Electric Irritability of Muscles.—Whilst the muscles of the thighs respond fairly well to the faradic current, the muscles of the legs, specially those just named, require a very powerful current, and respond very feebly. This is specially the case with the tibialis anticus in both legs. With the galvanic current very strong currents are required, and the polar reaction is the normal one,—that is to say, K C C is greater than A C C. There is thus no reaction of degeneration.

Cerebral and Mental Functions.—Intelligence is good, but he has a feeling of torpor, which often lasts all day, and gets worse at night. On trying to go to sleep he experiences a feeling as if he were dreaming, and he then feels as if he were going to faint. This causes him to rouse himself with a start, and so he often does not sleep all night, and never sleeps more than three hours on end. His memory, specially during the torpid states, is very much impaired, and he notices that he remembers events of his childhood more

easily than recent events,—that is to say, than events which have happened since the accident. He states also that his hand is very shaky, and causes him difficulty in writing.

Special Senses: Sight.—Since his accident this is impaired for distant objects; he cannot tell the time on the clock at the other end of the ward. For near objects his sight is good enough, but he says his eyes water greatly. On examination his pupils are found to react normally to light and in accommodation, and no changes can be detected with the ophthalmoscope. His other special senses are normal.

Locomotory System.—He can walk with the aid of a stick, but the feet hang down, and he has to raise them high to keep the toes off the ground. On standing with his feet together he sways considerably, but on shutting his eyes this swaying is not increased.

Alimentary System.—Teeth are bad; tongue fairly clean and moist; no tremors to be noticed. He has a sticky feeling in his mouth, and thinks there is less saliva than formerly. Appetite poor, and often feels thirsty. After taking food he is much troubled with pain in the stomach, and with flatulence. Used to vomit much after food, but not for the last ten days. Bowels confined, and has constantly to be taking medicine; says that if stools are not loose he has great difficulty in expelling them. Physical examination of the abdominal viscera shows nothing abnormal. Except that he suffers occasionally from palpitation, the circulatory system is normal, as is also the respiratory system.

Integumentary System.—His skin always tends to be moist, and he states that this is especially the case over the anæsthetic areas of the leg.

Urinary System.—Normal; no increased frequency of, or trouble in, micturition.

The diagnosis at which we have arrived is that there has been an injury to the vertebral column and spinal cord about

the lower dorsal and upper lumbar regions, and that this has resulted in gross changes in the cord at the part, and in less marked changes above. To understand this properly I shall ask you to consider with me—(1), the injury; (2), the changes in the cord at the part and below; (3), the changes in the cord and parts above.

(1.) The Injury.—This I believe to have been one of fracture with dislocation: you can understand that, locked together as the vertebræ are by their articular processes, these two—fracture and dislocation—almost certainly occur together.

Observe next that the locality where this fracture and dislocation have occurred is a very usual one, viz., about the last dorsal and first lumbar vertebræ. This you can understand when you remember the nature of the injury. The man was standing, stooping somewhat, when the weight fell on his shoulders and back. Now the dorsal vertebræ, together with the ribs, sternum, and intercostal tissues, form a sort of hollow cylinder, a very strong structure, and so can well resist a strain. Hence the effects of such will fall upon the nearest point in the comparatively weak and small column below, viz., the first lumbar vertebra.

[For the same mechanical reason you can understand how another part of the vertebral column, viz., the lower cervical vertebræ, are also specially liable to suffer from injury in a similar manner. This is the weak part immediately above, as it were, this strong hollow cylinder.]

As the result of this displacement the spinal cord may have been injured in various ways. It may have been pressed upon and narrowed by the lower fragment. There may have been some tearing of the dura, although this seems to be rare; there may have been hæmorrhage from parts around the cord, or, with more or less laceration of the cord itself at the part, there may have been hæmorrhage into its substance. Of whatever nature it may have been, it must have been a very severe lesion.

As further indicating the severity of the injury, we find that the patient was insensible for nine days, but that when he recovered consciousness, not only had he paralysis and anæsthesia from the seat of the injury downwards, with loss of power over bladder and rectum, but he had loss of power over his arms, and difficulty in breathing, lasting for about a month.

These latter symptoms, I believe, were due to concussion. We know that cases have occurred where paralysis and loss of consciousness followed injuries in which there had been no fracture or dislocation of the vertebræ at all, and in which, on post-mortem examination, no lesions of cord or brain could be discovered. In such cases we may suppose that as the result of the shock all nerve functions had been suspended,—just as, as Reynolds has put it, a piece of iron can be demagnetised by a heavy blow. In this case the shock must have been a specially severe one, abolishing his cerebral functions for nine days, and his spinal for about a month.

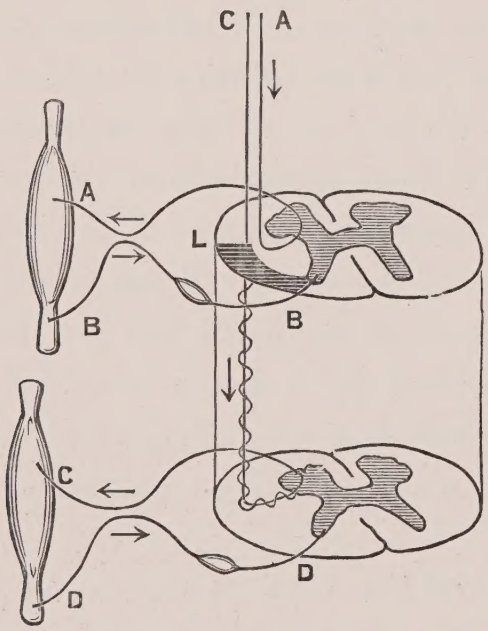
(2.) Changes in the Cord at the part and below.—It is certain that the injury to the cord which had caused abolition of function by shock above, must also have caused similar abolition below, and the return of control power over bladder and rectum after about one month's time might possibly be regarded as indicating that the lost power over these viscera had been due to shock rather than to direct injury to their centres and connexions in the cord.

It is almost certain, further, that as the result of the injury directly, or of a myelitis which is apt to follow such an injury, some sclerotic changes have occurred, and the question now is as to where these will be.

Are they in the grey matter? I do not think so to any extent. The bladder, rectal, and genito-spinal centres are intact, and the centres for the ankle-jerk and plantar reflexes are so also. Further, there is no muscle degeneration nor

tendency to bed-sores. Are they in the white matter, the connecting part of the cord? I think so certainly.

Looking at the subject theoretically, it is evident that we have to deal with a lesion which interferes with both motion and sensation, and probably the best way to explain this condition will be to invite your attention to the accompanying Diagram. This is intended to represent a longitudinal



section of the spinal cord. In it A A represent the tracts to the muscles of the thigh, with B B as the muscle sensory nerves corresponding, whilst C C represent the motor tracts to the muscles of the leg, with D D as their corresponding muscle sensory nerves. It is clear that a lesion in the region of the shaded area L would cause the motor and reflex phenomena which our patient presents, for it would leave his voluntary motor power over the muscles of the thigh unimpaired, and deprive him of voluntary motor power over the muscles of his leg; whilst it would at the same time explain the increased ankle-jerk and ankle clonus, and the absence of knee-jerk and knee clonus.

Similarly with sensation. To represent this diagrammatically a somewhat complicated diagram would be required;

but remembering that the skin sensory fibres do not remain long in the white matter, but having entered the grey matter pass from it back again into the white, and are conducted upwards in the posterior or lateral columns, we can easily imagine that a lesion in the position of the shaded area might cut the sensory fibres from the legs or thighs, and at the same time miss the sensory fibres from the feet. In our patient, as has been stated, sensation in the soles of the feet is not much impaired, whilst in both legs and in the front of the left thigh it is more or less abolished. Referring now to Gowers' table, the required portion of which I append,—

MOTOR.	(NERVES.)	MOTOR.	SENSORY.	REFLEX.
	L 1		1 Groin and Scrotum (front)	1
Lumbar muscles	2	Cremaster, Flexors of hip	2	2 Cremasteric
	3	Extensors of knee	3 Thigh: outer side, front, inner side	3 *Knee-jerk*
	4	Adductors of hip	4	4
Peroneus, 1. Flex. of ankle, Ext. of ankle.	5	Ext. & abduct. of hip	5 Leg, inner side	5 Gluteal
	S 1	Flexors of knee	1 Buttock, lower part	1 *Foot-clonus*
	2	Intrinsic muscles of foot	2 Back of thigh, Leg & foot except inner part	2 Plantar
	3	Perinæal and anal muscles	3	3
	4		4 Perinæum and anus	4
	5		5	5
	Co.		Co. Skin from coccyx to anus	Co.

we are disposed to put such a lesion in the cord somewhere about the cord-centres for the third or fourth lumbar nerves,—that is to say, about a point opposite the junction of the last dorsal and first lumbar vertebræ. Of course other possible explanations of the symptoms might be given, notably the possibility of some lesion of the "cauda equina," but we need not trouble ourselves further with what could be only hypothetical.

(3.) Changes in the Cord and parts above.—In the cord

above there are probably some changes of the nature of ascending sclerosis, but what I wish to draw your attention to now is his complaints of torpor, disturbed sleep, impaired memory, impaired sight, headaches, pains in the back and side, palpitation, etc. These are all symptoms which one meets with after concussions, and they are interesting, because they remind us of the train of symptoms which we hear of and see in the so-called cases of railway spine. As you know, after railway collisions it is common for a number of people, who of course have not been killed or have not lost limbs, but who have been simply "shaken," to develop such symptoms weeks or months after the accident, and presenting no objective signs of disease in brain cord, special senses, nerves, muscles or viscera, to declare themselves injured for life, and to claim compensation. Such individuals are apt to be looked upon as malingerers; but in our patient there is no inducement to malingering, and these symptoms are present. Their cause is not at all known. For the back pain, some irritation about the nerve roots has been suggested. It is evident that in an accident such as this man has sustained, the parts around must have been injured, hence we might expect pains here, as after fractures or severe bruises elsewhere.

As regards Prognosis.—The prognosis in injury to the spine is always better than in disease, as myelitis, lateral sclerosis, etc., and a further favourable point in this case is that improvement has occurred. But although I think we may expect some further improvement, I do not take a very hopeful view of the case. The presence so markedly of the so-called concussion symptoms seem to indicate that the severe injury has, as it were, taken so much out of him that we cannot expect him to present much recuperative power.

Treatment.—He is being treated by massage and electricity, galvanic and faradic, to his muscles. He is advised

to walk about as much as possible, short of fatigue. For the back pain we are trying the application of the faradic current, as we do in lumbago. Were it to become very severe I should have no hesitation in using the cautery. He is getting strychnia and nerve tonics internally.

A CASE OF FRIEDREICH'S ATAXY.

GENTLEMEN,—J. B., æt. 26, single, a soapboiler, was admitted to Ward 30 on October 17, 1893, complaining of weakness in the legs.

HISTORY : *Family.*—He is the fifteenth of a family of sixteen children, eight of whom died when young from causes unknown to him. Of the remaining eight, four are dead, two (sisters) of phthisis, and the others of causes which he cannot ascertain. His parents are both alive,—the father in good health, the mother suffering from rheumatism. No history of paralysis or of fits is in the family.

Personal.—Up till his present illness, which, according to his own account, began about six years ago, his health has been very good. He has never had syphilis. At work he has been, however, much exposed to sudden changes of temperature, and for the last seven years he admits having been in the habit of drinking to excess on Saturday nights. His present illness seems to have begun six years ago. At that time his speech became so affected that he had difficulty in making himself understood. According to his own account, this speech impairment came on rather suddenly, and without cause. After six months his speech seems to have improved somewhat, but has been since, and is now, markedly impaired. Three years ago he noticed for the first time that he was apt to stagger when walking. This was whenever he was in the dark, and he had then to guide himself with his hands in

order to prevent himself falling. Six months later he noticed that his hands trembled when he used them. The staggering in walking and trembling in the hands have both been gradually getting worse. He has been under treatment, in hospital and out of it, by massage, electricity, nitrate of silver, etc., but, he states, with no beneficial result.

Present Condition.—Height, 5 ft. 7½ ins.; weight, 9 st. 7 lbs. He used to weigh about 11 stones, but his general nutrition and muscularity are still fairly good. On examining him when stripped, some patches of chloasma were observed on the chest and abdomen, and a distinct lateral curvature of the spine was detected. On careful inquiry as to this, we found that it had probably been in existence about eight years. He himself had no idea that his spine was curved; but when we asked him how long his right shoulder had been noticed to be lower than his left, he stated eight years as the probable time.

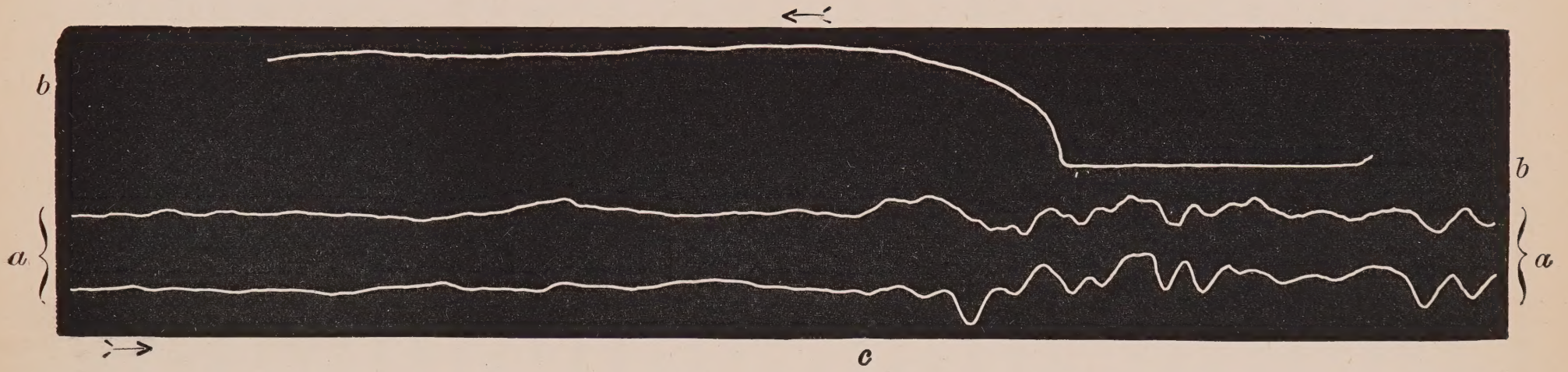
Nervous System: *Sensory Functions.*—At present there are no abnormal sensations except an occasional aching, not severe, in the loins. A fortnight ago he states that he felt for the first time a slight feeling of formication in both feet. Sensibility to touch, pain, and temperature is everywhere unimpaired. As regards special senses, sight is not impaired either for near or distant vision, and there is no diminution of the visual field in either eye. Colour vision is normal. The pupils are equal, and react readily to light and in accommodation. There is no paralysis of any of the ocular muscles, but when the patient is asked to look at an object placed to the extreme right or left there is some slight lateral nystagmus, affecting chiefly the eye that is directed outwards. With the ophthalmoscope no morbid changes can be detected. Hearing, smell, and taste seem normal. His muscular sense (sense of weight) is little, if at all, impaired, but his sense of locality is distinctly faulty. Thus, when with eyes closed he

is asked to bring his index fingers together, or to touch the point of his nose with one or other index finger, or to touch some point on one leg with the toe of the other foot, etc., he is invariably wide of the mark.

Motor Functions.—Voluntary motor power is rather impaired both in arms and legs. With the dynamometer the right hand registers 35 and the left 30, but whether the rather low grip-power is due to impaired conduction on the part of the voluntary motor nerve tracts, or to feeble musculature, it is difficult to say. This impairment of voluntary motor power seems to exist equally in arms and legs. The legs are, of course, much stronger than the arms, but proportionately their loss seems about the same. On testing the relative power of the flexors and extensors of the fingers and wrist, we found that in the former the power was about twice as great as in the latter. [Gowers says that "the flexors suffer more than the extensors, and the weakness in the flexors of the ankle may permit some degree of talipes to occur."] In our patient there is as yet no evidence of talipes. The electro-contractility in the muscles of his arms and legs showed no difference from that of health.

Reflexes.—The plantar and cremasteric are fairly well marked and equal. The abdominal and epigastric can hardly be elicited. The knee-jerk is absent on both limbs, and there is absolutely no trace of ankle-jerk or elbow-jerk or of clonus. As regards organic reflexes,—micturition, defæcation, generative power, respiration, and deglutition are normal, though he states that about a month ago he noticed temporarily a tendency to choke on swallowing hurriedly; this has not troubled him to any extent since.

Equilibration and Locomotion.—If he attempts to stand with his feet together and his eyes closed, he sways about for a little and then falls. It is worth while here to notice that it has been stated by some observers that the equilibration disturbance of Friedreich's disease is fundamentally



a Taxograph and *b* Dynamograph tracings, reduced one-half size.

Tracing *a* is from left to right, and the letter *c* shows the time at which the patient closed his eyes.

Tracing *b* is from right to left, and the patient was told to 'grasp' the dynamograph slowly.

different from that of ordinary tabes. Thus—here I quote from Ladame[1]—"Whilst in tabes closure of a patient's eye makes him lose his equilibrium, because he has no longer notion of the position of his limbs, in Friedreich's disease it is in consequence of choreiform instability. The patient sways whether his eyes are opened or closed; but in the latter case, having lost the control of vision, the swaying movements easily pass the limits of equilibrium, and he falls, although he may have preserved the notion of the position of his limbs." Of our patient I do not think this statement holds at all, and a tracing with the taxograph bears this out (tracing *a*). With his eyes open the lines are fairly even, and with them closed the undulations are exactly like those of an ataxic patient. In locomotion there is difficulty; the feet are lifted high, thrown forwards and outwards, and the heels brought down heavily on the floor. The gait is distinctly ataxic in character. When asked to walk backwards, he fixes his eyes on some object in front of him, and walks, however, better than we expected he should. When the patient is standing or moving about, a jerking and choreiform tendency can be seen in the arms when they are made to perform any movement. He can usually, however, when asked, hold his hands out quite steadily, but he has some difficulty in buttoning his clothes, in writing, or in picking up a pin. When standing on his feet or raising himself in bed his head is also seen to perform slight jerking nutatory movements.

Cerebral and Mental Functions.—Giddiness has always been a prominent symptom. His intelligence is good; he understands readily what is said to him. His memory is good, and he sleeps well. Speech is impaired; there is hesitation in getting out the words, but he says that this symptom is better than when he noticed it first, six years ago. The tongue, when protruded, shows some slight fibrillar contrac-

[1] *Brain*, vol. xiii. p. 467.

tions, but there are no tremors about the lips when he speaks. With the exception of the heart, which shows some increase of its area of dulness to the left, and the possible existence of a slight amount of aortic valvular mischief, the other viscera are perfectly healthy. [It is interesting to note that heart troubles are very common associates of Friedreich's disease, and this point, and the possible connexion between the morbid appearances in the spinal cord and the vascular and cardiac lesions, have been drawn attention to in an interesting way by Newton Pitt.[1]]

In this patient we have, I believe, an example of Friedreich's ataxy. At first sight, when we observed his ataxic gait in walking, his inability to stand with his eyes closed, and his absent knee-jerks, we for a moment thought of ordinary locomotor ataxy. But the absence of the characteristic initial lightning or girdle pains, of any impairment of tactile, temperature, and other sensations, of bladder trouble, of the Argyll Robertson pupil, and the presence of speech impairment and of jerking movements of the head and eyes, convinced us that it did not correspond with that disease.

We next thought for a moment of multiple cerebro-spinal or disseminated sclerosis. Certainly this disease, presenting great diversity in different cases as regards the amount of sclerosis and the localities where the sclerosis may exist, offers an anatomical explanation adequate enough for the symptoms which he presents. Some sclerosis of the postero-external column of the cord would account for the absent knee-jerks; some slight sclerosis of the direct and cross pyramidal tracts would account for any little loss of voluntary motor power in the arms and legs, and possibly also for the jerking character of their movements; a sclerosis of the cerebellar tracts in the cord and of their continuing fibres in the cerebellum would account for the equilibration disturbances and the giddiness, whilst sclerosis higher up in pons or

[1] *Guy's Hospital Reports*, vol. xliv. p. 392, etc.

crus would account for the impairment of speech, the jerking movements of head, and the nystagmus. But although it is interesting to note that by two such observers as Bourneville and Charcot, Friedreich's ataxy was likened to a combination of this disease and tabes, the clinical features are not those at all characteristic of multiple sclerosis. The speech impairment does not present the slow stacatto character, the nystagmus is not so marked, the movements of the limbs, when movement is performed, have a jerky character rather than the tremblings of multiple sclerosis, and there is an entire absence of the apoplectiform attacks seen in this disease. Again, the deep reflexes are lost in our patient, and not increased, as they are so markedly in cases of multiple sclerosis.

We have, therefore, concluded that the case is one of hereditary ataxy or Friedreich's disease, or, as Gowers prefers to call it, hereditary ataxic paraplegia. Now, here the first consideration which will occur to you is that to the term hereditary no great value is to be applied, because we have had no evidence of heredity in the case of our patient. This is certainly the case, and it would as certainly have made our patient appear to be a mere typical example of the disease had we been able to elicit evidence of heredity. But, as we remembered the other day when taking his case, it does not follow that the heredity or family tendency is not present. We can easily understand how a man in the position socially of an Infirmary patient may not be possessed of very complete or correct information as to the health of his relatives, and more especially when he has come in, as it were, at the fag end of a family of sixteen.

We have only to remember that, although this may not be altogether true here, in this disease the tendency to run in families is so well marked that the term hereditary has been applied to it. Perhaps the best illustration of this is the fact that sixty-five cases reported by one observer occurred in nineteen families.

As regards age, this affection shows itself at an earlier a e than does ordinary ataxy, the second dentition and puberty being about the commonest periods for it to appear. Our patient is now 26, but he gives six years ago as the time when the speech became affected, and eight years ago as the time when the spinal curvature was first noticed. As this spinal condition is due to muscular weakness, and may be regarded as a symptom of this disease, it would give 18 as the age at which it first developed in our patient.

As regards sex, all that need be said is that it seems rather more common in men; and as regards other points in the etiology, we have to remember that, being so distinctly due to congenital causes, a history of excessive exertion, exposure, alcohol, and syphilis, so common as etiological factors in other nervous affections, does not play such an important *rôle* in this disease.

The symptoms of hereditary ataxy as they have been described in other cases are markedly present in our patient. With absence of lightning pain or girdle pain, his cutaneous sensibility to touch, pricking, temperature, etc., are unimpaired, and with regard to his special senses nystagmus is the only abnormality. His knee reflexes are entirely absent, but his visceral reflexes—bladder and rectum—are absolutely normal. His equilibration in walking and standing is that of an ataxic patient, and the impairment of the co-ordinating power in his upper limbs is a prominent feature. Lastly, the jerking movements of the head and neck, the disturbance of speech, and the giddiness, which are all symptoms of Friedreich's ataxia, he presents in a marked degree.

The mode of onset of the symptoms in our patient presents some peculiarities. Ordinarily, though some nystagmus may be an early occurrence, the legs are first affected, then follow the arms, and then the speech disturbances. Our patient, however, states very definitely that his speech trouble was the first symptom, and that the legs and arms were not

affected till three or four years later. Further, as we have already seen, he told us that the lateral curvature of the spine had been noticed some two years earlier than his speech trouble. Now this spinal curvature, due as it is to weakness of the spinal muscles, is usually one of the later symptoms. Hence, as regards onset of symptoms, this case presents peculiarities. [In a case reported by Topinard, which Ladame regards as a typical example of Friedreich's disease, lateral curvature seems to have been one of the earliest symptoms, for in describing the case we read,—"Towards the age of 11 years, at La Pitié, M. Michon proposed an operation to remedy a sinking of the left scapula."]

As regards morbid anatomy, the principal deviations from the normal in such cases seem to be that, with the whole spinal cord somewhat smaller in section than usual, there is well-marked sclerosis of the postero-median columns, and sclerosis in varying intensity of the postero-lateral columns, of the cerebellar and ascending lateral tracts, and of the pyramidal tracts. That there should be sclerosis of these tracts we can easily understand from the evidence of the interference in these cases with the transmission upwards of those sensory impulses which are necessary for muscular co-ordination and equilibration. But that the sclerosis of the postero-median columns should exist, and exist so markedly, is noteworthy, for, as you know, the impulses of tactile impressions have been supposed to pass up in these tracts as well as those of muscular sensibility.

Inasmuch as in cases of Friedreich's disease tactile sensibility, and, indeed, cutaneous sensibility of all kinds, is unimpaired, this consideration would seem to indicate that the postero-median columns are, like the cerebellar and antero-lateral ascending tracts, more especially concerned in the transmission upwards of those impulses which are required for muscular co-ordination and equilibration.

Are we to believe that in this patient the pyramidal tracts are much affected? Here we have to remember that in similar cases in which opportunities for post-mortem examinations had been obtained, changes in these tracts were not very marked, although existent. I believe that in our patient they are not blocked to any extent. The voluntary motor power is still considerable, and the hand-grip with the dynamograph shows a fairly even line (tracing *b*). But I think it extremely probable, however, that in the ordinary progress of the disease the involvement of these tracts may become more marked, so that the affection may become a mixture of postero-lateral sclerosis and tabes, as Gowers has described it. Ordinarily, as you know, in cases of postero-lateral sclerosis the increased knee-jerk is an indication of the affection of the pyramidal tracts, but of course this help cannot be expected in Friedreich's disease.

The giddiness, nodding movements of the head, and the speech impairment indicate some—probably slight—sclerotic changes in the medulla and pons, and possibly also in the cerebellum, such as have been described in similar cases.

As regards pathology, it is believed that this disease is mainly due to an innate defective nutritive power. Its tendency to occur in families, and to show itself at an early age, before excesses of any kind have had time to bring about a premature wearing-out of the nerve machine, seem to indicate defective endowment rather than excessive expenditure.

On the question of prognosis all that need be said is that the disease is progressive, but slow, and may last for many years.

As far as we are at present able to judge in this individual case, it seems to be rather more rapid than usual, and the statement he vouchsafes, that he has lately at times felt some tendency to choke in swallowing, is of some importance

in connexion with possible involvements of parts in the bulb. The precise cause of this deglutition difficulty has been said to be an ataxy of the tongue.

For treatment, attention to the general health, massage, electricity, arsenic, phosphorus, and nitrate of silver have all been recommended. The tendency to contractions should also be remembered and guarded against.

References.

Brain, vol. xiii. p. 467. Dr P. Ladame.

Brain, vol. xv. p. 250. Dr Sanger Brown, with Discussion by Dr Ormerod and Professor Bernhardt.

Guy's Hospital Reports, vol. xliv. p. 369. Dr Newton Pitt.

ON A CASE OF

COMBINED PARALYSIS OF THE NERVES OF THE ARM DUE TO INJURY.

GENTLEMEN,—S. R., aged 24, a mason, born in and at present residing in Edinburgh, was admitted to Ward 30 on 7th December 1892, complaining of weakness of the left arm, the result of a fall.

History.—Family history good. Patient has been in the habit of taking occasional drinking bouts, but is, on the whole, moderate as to alcohol. Has always had a comfortable home, and never too hard work. Had measles in childhood, and whilst in Calcutta in 1887 had a slight attack of dysentery. Has had a few falls at his work, but none of them serious until the present one. This he met with whilst working at the window of a church in Newport, Rhode Island, U.S.A., on the 27th September last. From the window he fell a height of 12 feet, landing on his left shoulder and on the left side of his head. He states that when picked up immediately after the accident he was quite unconscious; that his left arm was found to have been folded under his body,—he was lying on his back; that his left ear and cheek were cut, but that there were no other serious injuries to his head. He was at once removed to the Newport Hospital. He regained consciousness some three and a half hours after the accident, and then discovered that he had lost completely

the power of motion and sensation in his left arm. Some four days afterwards he states that he noticed a gradual return of both, and he remembers that sensation returned first, and that the power of motion returned first in the fingers, and then in the hand and wrist. He states, also, that during the return of sensation his arm was so painful that he could not sleep at night, the pain being of a stinging character. He remained in the hospital for two weeks, and was treated with massage and electricity (faradic). After leaving he attended daily as an out-patient for six or seven weeks, but finding that the improvement in the condition of his arm did not continue, he came home to Edinburgh, and was admitted, as above stated, on 7th December 1892.

State on Admission.—Height, 5 ft. 6 ins.; weight, 9 st. 3 lbs.; well developed and fairly muscular. The left arm all over is slighter and feels more flabby than the right, and when asked to move the arm or forearm the loss of power becomes very marked. The flattening of the left shoulder caused by the wasting of the deltoid is specially apparent. There are no other obvious morbid appearances. The pulse and temperature are normal, and, with the exception of the condition of the left arm, he is a healthy, vigorous man. Let us now investigate more carefully this left arm.

Observe, in the first place, that the paralysis is associated with atrophy and absent reflex irritability of the muscles. Concerning this more will be said by-and-by, but at present this is sufficient to show that the lesion is either one of the peripheral nerves, or one of the cord involving the nuclei of origin of certain of the motor nerves in it.

Let us first consider the several muscles in detail.

Deltoid.—Its paralysis is shown by the fact that the patient cannot abduct the arm at the shoulder-joint, and its atrophy by the marked flattening of the shoulder.

Supraspinatus.—This muscle acts like the deltoid, hence when it is paralyzed along with the deltoid its loss of function

cannot well be demonstrated by a corresponding loss of movement. In this patient, however, its involvement is well seen by the wasting which it shows. It is worth mention that Gowers states that when the supraspinatus is paralyzed along with the deltoid, the head of the humerus falls away from the acromion far more than when the deltoid is paralyzed alone. This is illustrated in this patient to some extent. The head of his left humerus is distinctly lower than that of the right.

Infraspinatus.—This rotates the humerus outwards, a movement which this patient cannot perform, but the condition of this muscle is also best shown by the atrophy.

Teres minor.—This has a similar action to the infraspinatus, and is probably also involved in this case, although this cannot be demonstrated.

Subscapularis.—This rotates the humerus inwards, and is apparently *not* involved in this case. As you see, the patient, when asked, can rotate his humerus inwards, and you see also that the pectoralis major, trapezius, and latissimus dorsi are evidently unaffected.

The *rhomboids* are involved, as shown when the patient draws back his shoulders. The flattening in the left interscapular region is then well marked.

The *serratus magnus* is also involved. In this case its paralysis can best be seen when the patient tries, as it were, to push himself backwards with his hands. On the right side the digitations of the muscle can then be seen to stand out powerfully as compared with those of the left.

The *teres major* draws the humerus to the scapula, aiding the pectoral muscles. It is probably not affected in this case.

Triceps is not affected, as shown by its well-nourished condition and by its extensive power. When I flex his left arm and ask him to extend it, he does so with considerable force.

Biceps is markedly involved. He cannot flex the elbow, nor can he supinate the forearm when pronated.

Brachialis anticus is probably also involved, though this cannot well be demonstrated.

Supinator longus is certainly involved. This is well seen when he attempts to flex the elbow or to supinate the hand when it has been pronated.

The pronators and the extensors and flexors of the wrist and fingers seem all to work well. We were for a time not quite sure of the extensor carpi radialis longior, but we have now concluded that it is acting normally.

Next notice that these affected muscles show the reaction of degeneration. With the faradic current to their motor points we find no reaction on the affected side, whilst on the healthy they respond readily. The unaffected muscles of the left arm also respond readily to the faradic current. On the other hand, with the galvanic current to the affected muscles they respond quite readily, and the so-called "qualitative" or "polar" change is marked. That is to say, while

$$\text{KClC} \qquad \begin{matrix}\text{AClC}\\ \text{AOC}\end{matrix} \qquad \text{KOC}$$

is the order on the healthy side, we found that on the affected side there occurred in at any rate some of the muscles,

$$\text{AClC} \qquad \text{KClC.}$$

But this patient also presents some important sensory disturbances. There is, first of all, a well-marked area of anæsthesia over the deltoid muscle, corresponding with the distribution of the sensory fibres of the nerve to that muscle, viz., the circumflex nerve. Higher up on the shoulder, and over the supraspinatus muscle, is an area where anæsthesia is present, but to a less degree. Lastly, over the forearm there is an area of anæsthesia corresponding to the distribution of the subcutaneous fibres of the musculo-cutaneous nerve.

These anæsthetic areas can be shown very well by touching the skin over them, and noting where the sensibility becomes

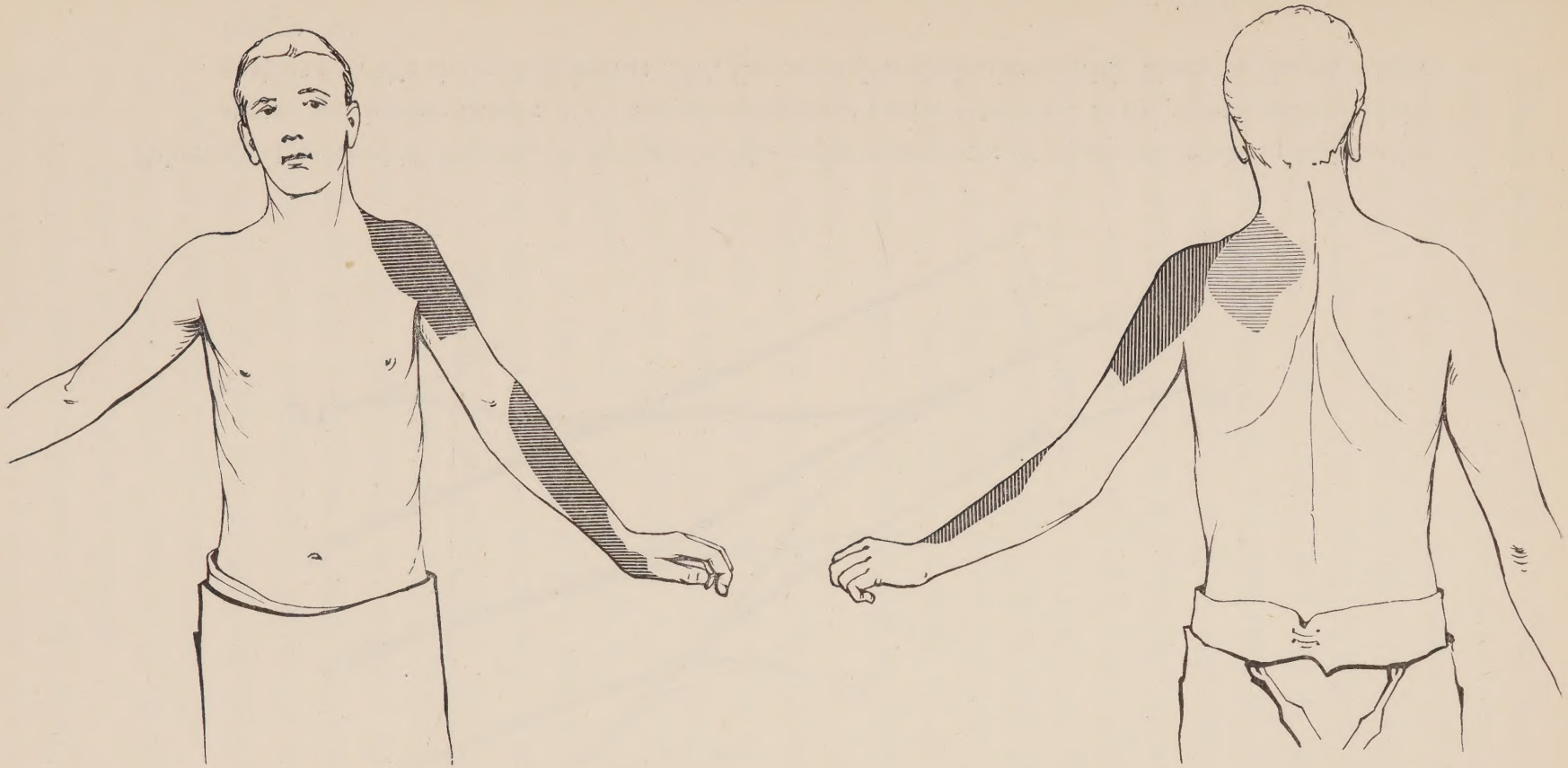
normal. They are well shown in the illustrations, which are taken from photographs. Thermal sensibility is also affected, as you see the hot and cold test-tubes applied to these areas cannot be distinguished.

Further, with the faradic current we find that whilst over the normal side the stimulus is felt when the approximation of the secondary to the primary coil reaches 15 mm., over the anæsthetic areas the stimulus is not felt till the approximation reaches 10 or 11 mm. With this diminution of sensibility to touch, temperature, and the faradic current, we find, further, a marked hyper-sensibility to painful impressions. Thus a pinch or a tap which, over the anæsthetic areas of his left arm and shoulder, causes the most apparent wincing, on his normal side has no effect whatever. There is anæsthesia with hyperalgesia.

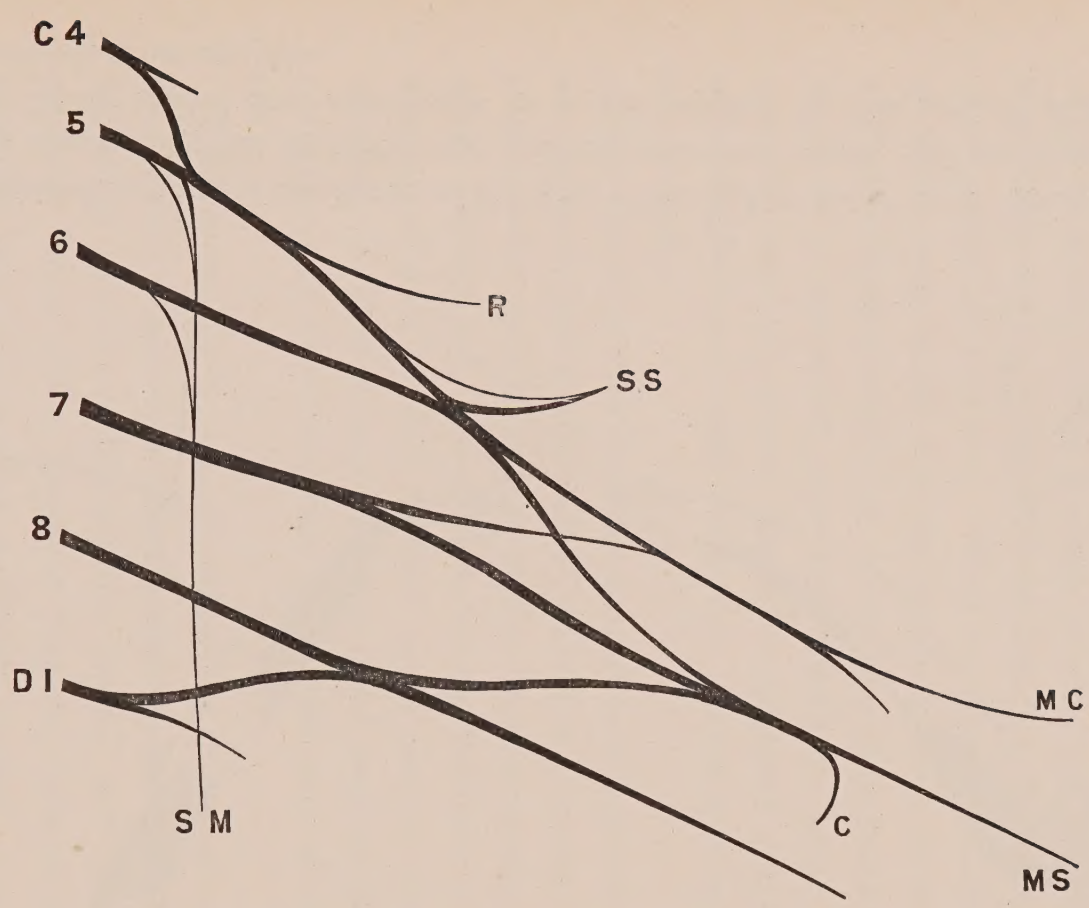
In connexion with these areas of diminished sensibility, we also endeavoured to discover if, with the galvanic current, an altered polar reaction occurs similar to what occurs in muscles when degenerated. In some such cases we know that a sensation is produced by ACl current more readily than by KCl current. In this patient we have found some evidence of this change,—only, however, over the deltoid area of the affected side.

Here, then, we have a case where, as the result of a fall on the shoulder some three months ago, we have paralysis of the following muscles:—Rhomboids, supra- and infraspinatus, probably teres minor, serratus magnus, deltoid, biceps, and probably brachialis anticus, supinator longus.

In addition, we have areas of anæsthesia over the shoulder, due to involvement of the sensory fibres of the circumflex, and over the radial surface of the forearm, due to the involvement of the sensory fibres of the musculo-cutaneous nerve. The area of impaired sensibility over the suprascapular region is probably due to involvement of some of the sensory branches of the cervical plexus.



Case of Combined Paralysis of the Nerves of the Arm.—The darkly shaded areas correspond with the parts supplied by the circumflex and musculo-cutaneous nerves, and are quite anæsthetic The lightly shaded area corresponds to a part supplied by the cervical plexus, and here anæsthesia is not complete.



Scheme of the Brachial Plexus.—S M, nerve to the serratus magnus; R, to the rhomboids; S S, to the supra- and infraspinatus; M C, musculo-cutaneous nerve, with fibres to the biceps and brachialis anticus; C, the circumflex nerve; M S, the musculo-spiral nerve, with fibres to the supinator longus, etc.

Thus, then, we have a lesion involving the nerve to the serratus, the nerve to the rhomboids, the suprascapular nerve, the circumflex, and certain of the fibres of the musculo-cutaneous and musculo-spiral nerves, viz., those to the biceps, brachialis anticus, and supinator longus and its sensory fibres. Noting that thus certain nerves and certain fibres are, as it were, picked out, we have to endeavour to find a locality where a lesion could possibly produce these effects.

In connexion with this, let me first draw your attention to the experiments of Ferrier and Yeo and the relations of various movements of the arm to the spinal nerve-roots. For example, on stimulating the 4th cervical root in the monkey, they found that the movements were elevation and retraction of the arm and flexion and supination of the forearm, by the rhomboids, supra- and infraspinatus, biceps, brachialis, and supinators.

On stimulating the 5th cervical root the movements were "similar to the last, but without retraction of the arm, and with extension of the wrist and first phalanges, by the deltoid, serratus, flexors of elbow, extensors of wrist, and long extensors of fingers." That is to say, that, with the exception of the extensors of the wrist and fingers, stimulation of the 4th and 5th cervical roots causes contraction of all the muscles which are picked out by the paralysis in this case.

Again, Gowers, in treating of the combined paralysis of the nerves of the arm, refers to the fact pointed out by Erb, that injuries to the neck sometimes cause a combined paralysis of the deltoid, supra- and infraspinatus, biceps, brachialis anticus, and supinators. On this subject Gowers goes on to say,—"Erb found that there is one spot between the scaleni, corresponding to the 6th cervical nerve, at which electrical stimulation puts all those muscles in motion."

Hœdemaker, who has described two cases of this palsy, finds the motor point in a line drawn from the sterno-clavicular articulation to the 7th cervical spine, 1·5 cm. from the edge

of the trapezius. The palsy is apparently dependent on disease of the 5th and 6th cervical nerves, and the 5th, it will be remembered, receives a twig from the 4th.

In our patient there is, as we have seen, involvement of certain sensory as well as motor fibres, and as the former of them, viz., those of the circumflex and of the musculo-cutaneous nerves, seem to have been traced to the 4th and 5th cervical spinal nerve-roots, I am inclined to think that these are the nerve-roots involved. From the fact that there is some appearance of change in the order of reaction of the sensory nerves to the galvanic current, it may, I think, be supposed that the spinal nerves have been injured outside the junction of the motor and sensory roots, and, of course, outside the ganglion on the sensory ones.

As regards prognosis, this is so far favourable. The nerve affection has been due to injury. It has not yet lasted too long to preclude recovery. The muscles have not lost their irritability to galvanism, some return of power has been noticed in the supination and flexion, and the supinator longus and biceps now show some tendency to respond to the faradic current. Further, the sensibility is improving.

Treatment.—This consists mainly in galvanism to the muscles and faradism to the affected areas of skin. Recently the faradic current has been also applied to the supinator longus and biceps. Massage to the affected limb is being applied daily. He is getting strychnia internally, and we shall use strychnia hypodermically if we think it necessary. To prevent stiffening at the shoulder he daily gives a little exercise to the joint by holding on by his hands to a horizontal bar which we have fixed up for him in the ward. Possessing good power in his fingers and hands, he can hold on quite easily, and the result has been that after about a week a threatened stiffening of the shoulder has disappeared.

A CASE OF PONS LESION.

GENTLEMEN,—The case which I am about to bring before you to-day is one which illustrates in itself some very interesting features, and which contains others which may be of value to you in the diagnosis of nerve lesions.

The patient is a man, R., 57 years of age, who was admitted June 20, and examined on June 21 and following days.

He complains of giddiness and difficulty in walking, and general weakness, of twelve months' standing; of numbness and loss of feeling on the left side of face, arm, leg, and trunk, of four months' duration; of inability to open the mouth widely, three months' duration; and of loss of power on the right side of the face, with double vision during the past seven weeks.

Family History is fairly good. His father died at the age of 55 of some chest complaint. His mother died of "old age" at 88. Three other members of the family died respectively from heart trouble, inflammation of the bowels, and small-pox. Others are alive and well.

Patient has had seven children, all alive and healthy. He has always had a comfortable home and good food, and is moderate in the use of alcohol.

At his work (a mason) he is often exposed to the weather.

Previous Health.—There is no history of syphilis. He seems to have been perfectly well till thirteen years ago, when after some heavy work and exposure to cold, he states

that he came home one night suffering from hoarseness of voice and uneasiness in the throat. Next day he went to work as usual, and the hoarseness became worse. At night his voice was gone entirely. He consulted a doctor, and his throat was painted and some local remedy applied, but after about a month, finding himself no better, he came to the Royal Infirmary, and was admitted to Dr Brakenridge's Ward.

His condition was diagnosed as paralysis of the left vocal cord, and it was thought probable that it was due to a small aneurism of the aorta involving the left recurrent laryngeal nerve. He was told to be careful in his living, and was sent out a little better. He comes back again to us after thirteen years, and, as we shall see, he still has paralysis of the left vocal cord.

From this time (thirteen years ago) he remained in good health until a year ago, when he began to be troubled with giddiness in walking, causing him to stagger as if drunk.

He noticed that he was obliged to keep his eyes fixed on the ground in front of him to prevent himself from staggering. The tendency to stagger was most marked towards evening; it was not so obvious in the morning.

As a result of this giddiness he once fell while stooping to lift a block of stone at his work.

He gradually became worse, and next he noticed that his general health was becoming somewhat enfeebled.

Shortly after this giddiness began—that is to say, about a year ago—he had an attack of vomiting which came on without any known cause. This lasted a week, was of considerable severity, and during it all food was rejected immediately.

Since then he has occasionally had slighter attacks of vomiting. They come on in the morning generally before, or it may be after, breakfast, and are preceded only by a slight feeling of nausea.

About this time also he noticed that he was troubled

with headaches, which, however, were not of a very severe form.

About four months ago he became aware of, as he describes it, a "numbness, tingling, and coldness, and sense of strangeness," on the left side of the face and body and on the left arm and leg, and a little later than this he noticed that in touching things with those parts he could not feel them so well.

A month after this he found difficulty in opening his mouth widely. His attention was drawn to this by the fact that he was obliged to use a smaller spoon than formerly.

Seven weeks ago he noticed a loss of power in the muscles of the right side of the face, and then he began also to see double, indicating a paralysis of some of the eye muscles. With the paralysis of the muscles of the right side of the face he noticed that his right eye did not close properly. He says he noticed this, because, when lying in bed with his eyes shut, as he supposed, he still saw light.

He states also that the vomiting and the feeling of weakness have been getting worse during the last four weeks.

He was seen about a month ago, prescribed for, and told to return in a month. This he did, and was admitted to Ward 30.

Present Condition.—The patient is a man 5 ft. 6½ in. in height, and at present weighs 10 st. 4 lbs., but he used to be about 12 st. when in health. His muscles are soft and flabby. The right half of his face is absolutely motionless. There is no wrinkling of the forehead, and no reflex closure of the lid on touching the conjunctiva of the right eye. The eye is widely opened, and the conjunctiva is slightly inflamed. The lower lid is also a little removed from the globe, but not much.

There is also evident hollowing in the right temporal fossa, due to paralysis and wasting of the right temporal muscle.

Alimentary System.—The tongue is not in any way paralyzed, but can be moved freely in any direction. He

complains of dryness of the mouth, especially on the right side. This is important, because with the portio dura, as you know, you often have the chorda tympani involved, and so salivation affected.

During mastication the food tends to collect on the affected side of the mouth in the hollow of the cheek, and he has to clear it out with his finger. The palate shows some indications as if its right side were a little lowered, but this is not very well marked. There is no difficulty in swallowing.

The bowels are very constipated.

Physical examination of the abdomen reveals nothing worthy of note.

Hæmopoietic System is normal.

Circulatory System.—Pulse 88, strong and full. The vessels are a little thickened, but there are no indications of aortic aneurism.

Respiratory System.—There is a slight cough and spit. The left vocal cord is quite paralyzed, as already stated.

Integumentary System.—This is normal, except that there is a slight tendency to œdema of the legs.

Urinary System.—Patient has generally to rise once at night to pass water. The act of micturition is at times slightly delayed, not sufficiently to be of importance.

Sp. gr. of urine, 1020; no albumen; no sugar.

Nervous System (*Sensory*).—Patient complains of coldness, numbness and tingling, as if the parts were "asleep," in the left arm and leg, and on the left side of the body and face. This is specially marked on the arm and front of the chest. There is some impairment of feeling all over these parts. The area of incomplete anæthesia is limited by the middle line of the body. There is no hyperæsthetic part.

On the right side sensation is perfectly normal, at any rate as regards the trunk and limbs. On the right side of the face there *seems* to be a little hyperæsthesia, but it is difficult,

if not impossible, to determine this, since we have only the defective side to compare it with.

Sensation of locality is impaired on the left side. He cannot say exactly where you have touched him.

There is also some impairment of sensation in the left conjunctiva and in the mucous membrane of the nose and cheek on the left side. Ammonia held to the nostrils stimulates the right more than the left.

Sensibility to weight is not affected.

Temperature sense is impaired on the left side also. Cold is often mistaken for hot, but hot is never mistaken for cold.

Sensibility to electric current on the left side is also diminished, and also sensibility to pain.

Rate of nerve conduction on the left side is diminished.

Muscular sense on the left side is not much affected.

Special Senses: Smell.—The sniffing movement of the right nostril is not well marked, but he smells such a substance as clove oil equally well on each side. As just stated, ammonia is perceived better on the right side, not so well on the left, since it stimulates the nerves of common sensibility.

Hearing.—He hears constant noises in the right ear, and loud sounds are sometimes painful. But the ticking of a watch is not heard so well with the right ear as it is with the left.

Sight.—The right eye points over to the left, and is completely paralyzed with respect to the external rectus.

The left eye moves well enough to the left, but only a little to the right. Hence there is modified paralysis of the internal rectus of this eye.

Dr Sym examined the eyes, and the following is his report:—

"There is paralysis of the right movement of both eyes. It is rather a paralysis of movement than of muscles. The internal rectus of the left side makes no effort to con-

tract when the patient is asked to look at an object moving to the right, but it feebly contracts when he is told to converge for a near object. The other muscles are free. There is homonymous diplopia. Fundus seems healthy. Disc is a little grey. There is no appearance of former neuritis. Field of vision is fairly good. Pupils are of medium size, and contract to light and accommodation. This erroneous projection leads to great giddiness in standing or walking."

Taste.—We have had some considerable difficulty in satisfying ourselves as regards the condition of taste in this case. In the first place, the patient cannot open his mouth very widely, so it is difficult to get at the back part of the tongue. In the second place, we have to contemplate the possibility of the existence of lessened common sensibility on the left half of the tongue, and of this causing some confusion. But all our observations go to indicate that on the whole of the right half of the tongue, front and back, there is a diminution both of taste and of common sensibility. When, for instance, a little quinine, or sugar or salt, is applied to the tongue, or taken into the mouth, he distinctly tells us that he feels the taste only on the left side, and all over it. Similarly, when the tongue is touched or pricked with a needle, he tells us that he feels it distinctly better on the left side. This is not altogether what we should have expected, but it certainly is what he says he feels.

Motor.—Organic reflexes are quite normal. Skin reflexes do not in any way deviate from health, but the conjunctival reflex is, of course, absent on the right side.

With regard to the voluntary muscles, there is nothing very noticeable, except with regard to the muscles of mastication. The masseter and temporal are paralyzed and atrophied on the right side, and they, with the facial muscles, show the reaction of degeneration. As regards the pterygoids, we find that the lower jaw is moved slightly over to the

right side when it is depressed, and that the patient cannot so well perform a lateral movement of it to the left side. This indicates paralysis of the right external pterygoid muscle.

Co-ordination.—The patient performs movements, such as buttoning his coat, perfectly well.

He can stand with his eyes shut and his heels together without swaying. When walking with his eyes open he tends to go to the left, and in attempting to correct this he staggers. He has great difficulty in going up and down stairs, on account of the confusion arising from his double vision.

With one or both eyes shut, he walks much better, and can even walk backwards fairly well.

So much for the symptoms and morbid appearances. What we have to do now in connexion with the consideration of the case is to make up our minds first as to the locality, and secondly as to the nature of the lesion.

Let us take first of all the paralysis of the face. This paralysis of the face, you can see at once, must be due to a lesion of the nerve itself, or of its nucleus. It is not due to a lesion above the nucleus.

We are inclined to think that it is the *nucleus* of the facial nerve that is affected.

I think with these rough diagrams (see pp. 152 and 153) you will be able to follow me.

The nucleus of the portio dura or facial nerve (F) lies in the lower part of the pons Varolii, and we believe that we have a lesion somewhere at the lower part of the pons involving this root. But a lesion involving the root of the facial must be quite close to the root of the sixth nerve (VI.), and it is very likely to involve it as well, and this would explain the paralysis of the right side of the face, and the paralysis of the external rectus of the right eye.

But what about the left eye? This may be explained by the connexion of the sixth nerve on one side with the third nerve on the opposite side, so that such a lesion is apt to

interfere with the co-ordination of the sixth and third nerves. Dr Sym's report, as we have seen, indicates this as the explanation.

Further, a lesion here will also be very near the motor nucleus of the fifth (Vm.) nerve which supplies the muscles of mastication. Hence the paralysis of these muscles can also be explained.

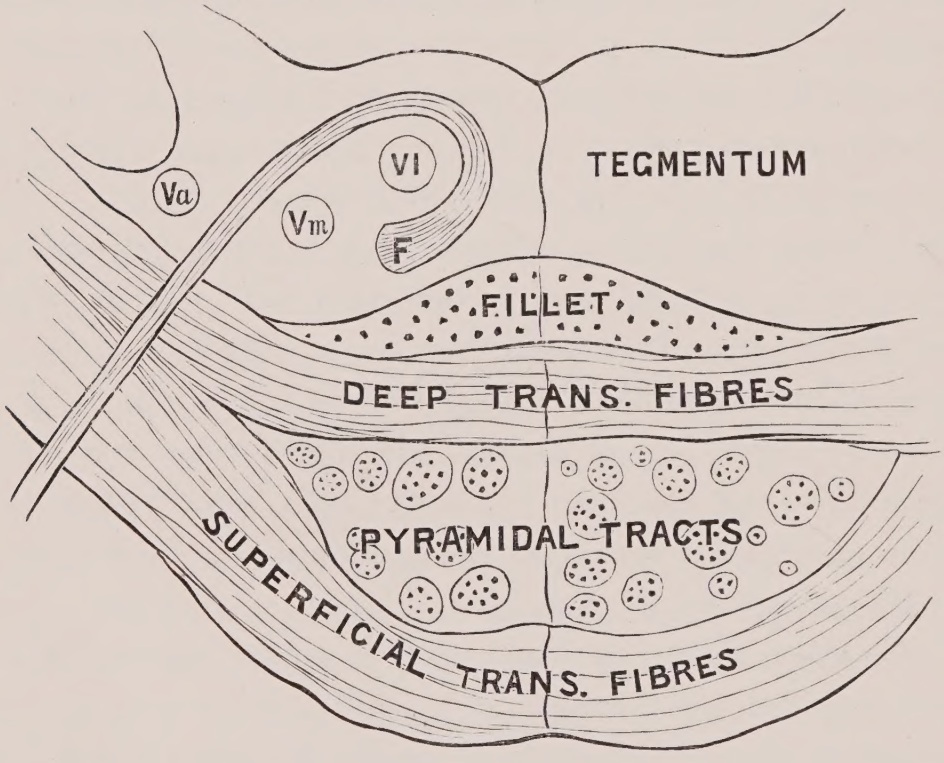


Diagram of section of the lower part of the pons, showing the connexion of the facial, sixth, and motor fifth nerves (mastication).

F. Facial nerve. *VI.* Sixth. *Vm.* Motor of fifth.

As regards the giddiness. Notice that this may be explained as being due to the disturbed working of eyes. It may be entirely due to this, but it is possible that it may be due in part also to some of the fibres of the portio mollis being involved. Thus we know that from a little lower down than the positions of the facial, sixth, and motor fifth nuclei the portio mollis or auditory nerve arises, and we know also that in connexion with it there are two nuclei of origin. We know also that whilst one of these nuclei seems to be mainly

connected with fibres from the cochlea, and so to be concerned in hearing, the other is mainly connected with the semicircular canals and vestibule, and is supposed to be concerned in equilibration. In the diagram they are both indicated by the area marked Au, and it is evident that a lesion involving the facial, the sixth, and the motor fifth is very likely to involve this nucleus also. In this way, therefore, might be explained in part the giddiness. No doubt it and the equilibration disturbance are largely due to the eye conditions, but he tells us that even when he sits quietly with his eyes closed he still feels the giddiness to some extent.

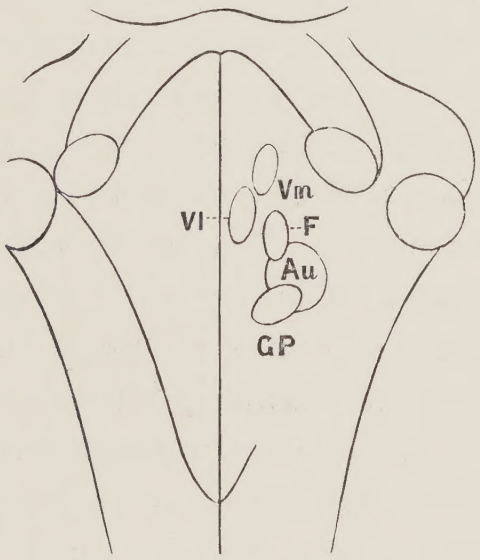


Diagram showing the relative position of the nerve nuclei below the floor of the fourth ventricle.

F. Facial. *VI.* Sixth nerve. *Vm.* Motor of fifth.
Au. Portio mollis. *GP.* Glosso-pharyngeal.

Hearing.—As you are aware, in paralysis of the facial nerve hearing is often affected owing to the action of the stapedius muscle being interfered with. In this case we have also, as just stated, to take into consideration the possibility of the auditory nucleus itself being involved.

Next, as regards the loss of sensation on the left side of the head, trunk, and left arm and leg.

The sensory nerves from the left leg, arm, and side of

the trunk pass into the posterior part of the cord, and having decussated either in the cord or in the medulla, pass through the tegmentum of the pons, up the crus to the brain. We can understand, therefore, that a lesion here may affect these and account for the hemi-anæsthesia.

Further, to account for the impairment of sensation on the skin and mucous membranes of the left side of the face, we have to suppose that the lesion on the right side of the pons, while leaving unaffected the sensory part of the fifth nerve on that side (*Va*, diagram, p. 152), is in one way or other interrupting the continuity of the fibres which convey impulses from the sensory part of the fifth on the left side, upward to the right side of the brain.

Taste.—As regards taste, we have seen that in our patient not only is this special sense impaired over the right half of the tongue, but that common sensibility appears to be also impaired in this situation. The loss of taste is explained by the view that the glosso-pharyngeal is the sole nerve of taste, and that it supplies the back of the tongue through its main trunk, and the front part through the *pars intermedia* and chorda tympani. Hence in our patient we must suppose that the glosso-pharyngeal nucleus or fibres of origin are involved (diagram, p. 153).

But how can we explain the impairment of common sensibility on this right half of the tongue with no other evidence of lesion of the sensory fifth nerve on that side? Explanations might be hazarded, but they could only be conjectural.

Larynx.—As regards this the question might be asked, Can the paralysis of the left vocal cord—which was noticed thirteen years ago, and which is now very much as it seems to have been then—be a part of his present disease? I believe not. Neither do I think that it is due to aneurism. Probably it is due to some inflammation of a rheumatic nature, which has led to adhesions between the left arytenoid and parts around.

As regards locality of the lesion in this patient, therefore, I believe it to be situated in the right side of the lower part of the pons, involving the nuclei of origin of the facial, sixth, and motor fifth nerves, extending downwards so as to implicate to some extent the nuclei of the glosso-pharyngeal and portio mollis, and upwards so as to implicate the sensory fibres which have crossed from the left side of the head, trunk, and limbs.

Nature of the Lesion.—This is obscure; but since the condition has been going on steadily for twelve months, I think that probably it is of the nature of an infiltrating glioma. It is of slow growth, and although there is some headache and vomiting, there is no distinct optic neuritis. Still I think it is of this character.

Treatment.—It is not necessary to say much about this. We are giving him the iodide and looking after his general health. This is all we can do at present.

CASE OF ADDISON'S DISEASE.

GENTLEMEN,—A. C., aged 52, an upholsterer, born and residing in Edinburgh, was admitted to Ward 30 on January 27, 1893, complaining of great weakness and brown colouring of his skin.

History.—Family: father died of inflammation of the lungs at the age of 39, and from the patient's account this seems to have been acute pneumonia. Mother died at 83, and was always a strong, healthy woman. He has one sister only, and she is rather weakly, although evidently not phthisical. Patient is married, and has been the father of a family of seven, of which four are dead,—one at the age of 6 years, of water on the brain.

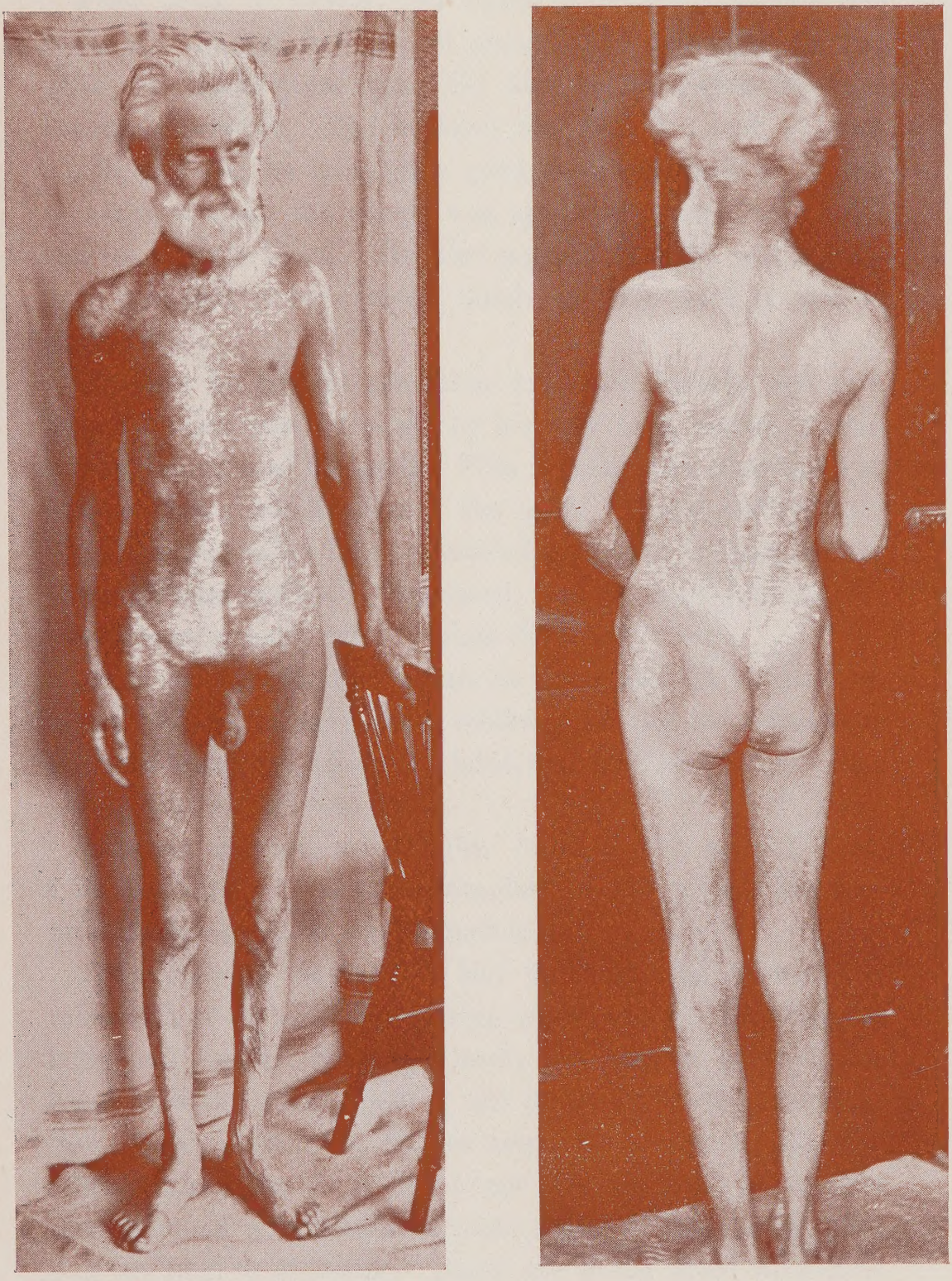
Habits as to Food and Drink.—He gives a strong alcoholic history, and states that he has also taken tea in excessive quantities.

General Surroundings.—At home he has only one room for himself and his family, and he says his house is damp. At work his surroundings are also unfavourable. He is much engaged making mattresses, and so is constantly in a very dusty atmosphere. This, he says, makes him cough, and causes his spit to become black for weeks at a time. His average day's work is nine hours.

Previous Illnesses.—Had measles when a child. He says he is very liable to catch cold, and that he always gets a cough when winter comes on. He gives no history of venereal disease.

CASE OF ADDISON'S DISEASE.

Portion of the Anterior Abdominal Wall, showing at parts the arrangement of the spots in rows and their ring-like character.



CASE OF ADDISON'S DISEASE.

Time, Mode of Origin, and Cause of Present Illness.—He states that he was feeling well enough, and was quite fit for work till three years ago. He then noticed that he was becoming weaker. He noticed, too, that he had a hard cough, and that he sweated a good deal, especially at night. Sometimes, when the cough was specially severe, he would spit up a little blood. He also noticed that he was losing flesh, and that if he sweated much his skin used to itch a great deal.

He kept on in this condition until five months ago, and then attention was attracted by his wife to a brown coloration at the back of his neck. This discoloration was uniform over an area about the size of the palm. It was not spotted, but a few days after this he noticed a number of spots on his chest. These gradually increased, and he next noticed the legs becoming coloured, and then the arms.

Since the discoloration began, he states that he has noticed himself becoming very much weaker; he has been troubled at times with pain across the loins, and his stomach has given him a good deal of trouble.

State on Admission.—Height, 5 ft. 7 in.; weight, 8 st. 1 lb., used to be 9½ st.; his muscles are flabby, and his face presents the appearances of anæmia.

Integumentary System.—When stripped, the brown discoloration of the skin can be seen widely distributed over the body and limbs. Over the neck, the greater part of the trunk, and the greater part of the limbs the brown discoloration is fairly uniform, but at some parts, and notably the anterior abdominal wall, this is not the case; but its appearance is one of a multitude of spots, round in shape, and about ⅛ of an inch in diameter. These are much more numerous on the left side of the lower part of the abdomen, where they are arranged as if in regular rows right up to the middle line, where they abruptly cease. These spots can be made out very distinctly over the other parts of the trunk and limbs;

but, of course, where they have become so numerous as to merge into one another, the appearance of the parts has changed.

If the spots are examined more carefully, they are in many cases seen to be ring-like,—that is to say, to present a dark-brown margin and a lighter-coloured centre. In many cases, too, their centre shows a hair, whilst in other cases the spots seem to encircle a sweat-gland. This latter point we have satisfied ourselves upon by examining them carefully after he has been sweating, for then many of them show, in their lighter-coloured centres, a raised and somewhat sodden appearance of the epidermis.

As previously stated, the face, though somewhat sallow, does not show the pigment; but the darkening is fairly well marked in the external genitals. The axillæ and the nipples cannot, however, be said to be more pigmented than the surrounding skin. Over the non-discoloured areas, specially of the abdomen, the skin appears to be—and in this we are corroborated by the patient's own statement—rather whiter than normal, and on each arm small spots, about half an inch or so in diameter, of leucoderma can be seen. The palms and soles show no pigmentation. Over the outer aspect of the left elbow joint there is an area, oval in shape and about one inch in length, where the skin is slightly reddened and not pigmented. The patient tells us that the skin had been browned here; but that a few weeks before his admission he had knocked his elbow against a wall and skinned it at the part, and that when he took the scab away he had noticed it white.

The hair of the patient's head and beard is uniformly white, but he says that he began to turn grey at the age of 36 years. He believes, however, that the marked whiteness has existed only for the last three years,—*i.e.*, since he has noticed his illness coming on.

On examining the hairs of the body, notably about the

pubes, we noticed that on the left side, or wherever the pigmentary coloration of the skin is more marked, the hairs also seem to be more pigmented. This is not always the case, for though on close examination we observe that the hairs emerging from the centre of brown spots are more frequently brown than those emerging from a non-coloured area of skin, we find many brown spots giving origin to white hairs, and many pale areas of skin giving root to brown hairs.

The nails in our patient show, as is usual in Addison's disease, no discoloration ; but they are markedly thickened, specially those of the thumbs, and index, middle, and ring fingers. The toe nails are apparently unchanged.

As regards the mucous membranes, the lips show patches of brown pigmentation, mostly along their line of contact; the hard palate shows also patches most markedly, and the inner surface of the cheeks to a less extent. The mucous membrane of the tongue does not appear to be affected.

Respiratory System.—With the history of cough, of night sweats, and of slight hæmoptysis, we carefully examined the chest, and found some impairment of the percussion-note at the left apex posteriorly, with slight harshening of the respiratory murmur, prolongation of the expiration, and increase in the vocal resonance. The at present scanty muco-purulent and rather frothy spit has been examined, but no bacilli have been found. It shows however, curiously, the pneumococcus.

Circulatory System.—When at rest he has no symptoms of circulatory disturbance, but if excited he is liable to palpitation, and though he never has fainted, he is apt to feel faint on making any sudden movement. Dyspnœa, also, he does not complain of, except as the result of exertion.

Physical examination shows the heart to be slightly enlarged (dilated), its left border being a little outside the nipple line. With the exception of a hæmic bruit at the

apex, and a slight "bruit de diable" in the veins of the neck, there are no murmurs.

Pulse is 110 per minute, very soft and compressible, although regular, and the low tension is shown well by the sphygmographic tracings.

The blood when examined showed the red corpuscles to number 3,500,000 per cmm., and amount of hæmoglobin to be 45 per cent.

Dr Muir has kindly examined some specimens of it for us, and his report is as follows :—

"The red corpuscles show considerable variations in size, many being undersized. There were also a few irregularly-shaped corpuscles. The corpuscles are pale, and form *rouleaux* somewhat imperfectly. The blood-plates are markedly increased in number. The leucocytes are also somewhat increased, specially the large variety.

"The blood thus shows nothing characteristic, the changes being such as are generally met with in cachectic conditions."

Alimentary System.—His appetite is fairly good, and he is rather thirsty, but he has now—*i.e.*, since his admission—no vomiting. He used to suffer from vomiting and pyrosis. His bowels are usually confined; and he says that every fortnight or so he is apt for two or three days to be attacked with vomiting and diarrhœa. He says also that he sometimes takes a rather severe pain in the epigastrium.

Physical Examination reveals no enlargement of liver or spleen, nor any other abnormality.

Genito-Urinary System.—Urine: Quantity has varied between 36 and 80 ozs., colour rather pale, reaction acid, specific gravity 1014 to 1020, no albumen nor sugar. On several occasions we have estimated the quantity of urea, but found no marked decrease. Neither have we found any increase of indican.

Hæmopoietic System.—Some slightly enlarged glands in the groins and axillæ can be detected.

Such, then, is our case. We have concluded that we are dealing with an example of Addison's disease,—of a disease in which some morbid condition of the suprarenal capsules is the main lesion. But our patient presents some rather peculiar and very interesting features, which I wish now particularly to draw your attention to.

In the first place, as regards etiology. This disease is, of course, more frequent in men, but notice that our patient is rather older than the average. Next, as you will remember, the most frequent disease of the capsules in this malady is a tubercular one; it is interesting to note that although in his family history there seems to be no very marked phthisical tendency, he has had a cough and hæmoptysis. A point which has been adverted to by some of those who have written on the subject of Addison's disease is, indeed, that the disease is not more common in the phthisical.

Another factor in its etiology which has been instanced is injury to the back. In our patient we have, however, no evidence of this, but he has had to do hard physical work in unhealthy surroundings.

As regards symptomatology, the first point to which I wish to direct your attention is, that in our patient the one special symptom of the disease, viz., the debility, preceded the other, the bronzing of the skin. He tells us that he has been feeling weak and ill for some three years, whilst that the bronzing of the skin was not noticed till some four months ago. In the *Croonian Lectures* for 1875 Dr Greenhow states that the number of cases in which the bronzing is preceded by the debility and in which the debility is preceded by the bronzing are about equal, and cases seem also to have occurred where these two main symptoms were noticed to have occurred together.

The localities where the pigmentation shows itself is also important. As you are aware, it tends for the most part to

affect first and most markedly the exposed parts—face, neck, and hands, and also those parts which normally contain most pigment, viz., the axillæ, nipples, umbilical region, external genitals. As you will see, this is not altogether the case with our patient. Although he reports the coloration as having been first seen on the neck, you will see that it, with the face and hands, is not so dark as the trunk at many parts, and that though the external genitals are pigmented, the axillæ, nipples, and umbilical region are not specially so. As is usually the case, the palms of the hands are distinctly less coloured than the dorsal surfaces, and although the finger nails are thickened in the manner already described, we can see that their matrices are not discoloured.

Another point worthy of note is that abrasions of the skin may be followed by an excess of, or by an absence of pigment. As you will remember, our patient has a small spot of a pale colour on his left elbow which has followed an abrasion, and he distinctly tells us that before he received this injury the skin was dark in colour.[1] It would seem as if the absence or excess of pigment depended on the depth of the injury. Where this has been deep the resulting scar will be white; where superficial, it will be much darker than the surrounding portions. Examples of the latter have been quoted following the application of a blister, and similar darkenings have been observed to follow the slight skin irritation caused by garters. A case very interesting from this point of view was reported by Mr Nicholson in 1872, of a baker lad who presented on his shoulders dark stripes, corresponding to the bands by which the basket he carried was slung at his back.

The transition from the darker to the lighter areas of skin is also to be noticed. As a rule it may be said that this is never abrupt, the darker merging gradually into the lighter

[1] Since his admission we have noticed fresh brown spots appearing over this whitened area.

apparently normal areas. An exception to this is met with, as is often the case, where there is leucoderma, as shown well in plate XI. of Addison's monograph, and the small spots of leucoderma on our patient show well a similarly abrupt margin. Still at many parts a merging of the dark into the normal coloured areas can be made out.

But now let me draw your attention to the peculiar spotted appearance which in our patient this discoloration presents. As already stated, he tells us that the first discoloration noticed was on the back of his neck, and that this was a large, uniformly diffused patch. Over the other parts of the body and limbs the pigmentation seems to have begun as spots rounded in shape, and by gradually becoming more numerous, merging into one another to produce a uniform browning. The occurrence of spots to some extent is not unusual in Addison's disease. In one of the plates (X.) of Addison's monograph several such are figured, and Greenhow describes the "appearance of small, well-defined black specks, like black moles or freckles, on already discoloured portions of skin." It is evident, however, that the spots in our patient, many of them presenting the appearance of small rings with pale centres, are different altogether from what Addison has figured and Greenhow has described.

The arrangement of these spots, specially on the lower part of the left side of the abdomen, is very curious. As has been stated, some seem to be connected with hair follicles and others with sweat glands. Their curious distribution suggesting some nerve influence, we have tested the skin sensibility to touch, pricking, and temperature, comparing the spots with the surrounding skin, but have been unable to detect any difference.

As regards the mucous membranes, the appearances are the usual ones of Addison's disease. The conjunctivæ are white, the lips show pigmentation mostly at the line of contact of the one with the other, and the hard palate and

inner surface of cheeks show several small dark-coloured areas.

As regards the epidermal appendages, the thickening of the thumb and finger nails has been already referred to, and we have already also referred to the tendency for the body hairs to be dark when emerging from a pigmented, and blanched when from a non-pigmented spot.

Further, the general symptoms,—the extreme debility and sense of weakness, the lumbar and epigastric pain, the dyspepsia and vomiting, the feeble and somewhat quickened pulse, all contrasting with the patient's showing no great emaciation and no great loss of subcutaneous tissue,—are just what we expected in such a case.

Since he has come in our patient has improved very much in condition, as the result probably of rest. He has gained weight (6 lbs.), he feels much better, and his pulse is now about 70, and of much better tension. In this connexion we have to remember that, although the disease is practically uniformly fatal (one case of recovery only is reported), remissions are frequent, and it may last for years. In these remissions the general health improves; and, what is more interesting, the skin-coloration lessens. I cannot help thinking that a lessening of the colour has occurred to a slight extent in this case.

As regards the modes of death in such cases. This is usually by asthenia, the mind remaining clear to the last in spite of the tremendous nervous prostration. Sometimes, when so very weak, a syncopal attack ends the scene, and in other cases death is preceded by coma. It is interesting to know that in one example of the latter mode of termination acetonuria was present, and it is important to remember that, like diabetes, this also is a disease which may be treacherous. Greenhow gives several examples of Addison's disease in which severe constitutional symptoms developed suddenly, and proved fatal in a few days.

The treatment of the disease is closely bound up in the pathology, so concerning this a few words are necessary.

As you are aware, this association of intense prostration and bronzing of the skin was shown first by Addison to be connected with disease of the suprarenal capsules. Since Addison's time much work has been done in connexion with the subject, although we still have a great deal to learn.

We know, however,—

(1.) That in this disease the pigment is deposited mainly in the cells of the rete, as is the case in the dark races of man, but that it may be found to some slight extent more deeply in the fibrous tissue of the cutis.

(2.) That although in patients suffering from cancer, tubercle, lymphoma, etc., and in whom the suprarenal capsules have been unaffected, areas of skin pigmentation are not infrequent, such cases are to be regarded as being altogether distinct from Addison's disease.

(3.) That it has been observed that with diseased capsules there may exist the extreme debility without the bronzing of the skin, as well as the extreme debility with the bronzing. Such instances have been explained on the theory that in the former set of cases the medullary part of the capsules, *i.e.*, the part which contains the nerve structures, is alone involved, whilst that in the latter set the cortical part, *i.e.*, the part concerned in pigment formation, is also diseased. In all the cases, further, in which prostration alone existed, it is reasonable enough to suppose that, had the patients lived long enough, bronzing might also have supervened.

(4.) That the semilunar ganglia are more or less involved in suprarenal capsule disease, and that they may have to do with the induction of the severe nerve symptoms.

We know, also, that the affection of the capsules is usually a tubercular one; that with other affections of these bodies, *e.g.*, cancer, pigmentation is not so frequent; but we know, too, that cases of Addison's disease have been seen where the

capsules seemed to have undergone atrophy, where, as has indeed been stated, the capsules had undergone the same change as does the thyroid in myxœdema. In such cases we can conceive that the administration of the suprarenal capsule of a sheep or ox would be as efficient as is that of the thyroid of such an animal in myxœdema. If, on the other hand, there exists tubercular or other disease of the capsule, we cannot, of course, expect anything like so much.

Up till now our patient has been treated only by rest, diet, and tonics, and, as you see, there has been very considerable improvement.

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